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CONTENTS

Environmental factors in relation to snap-bean diseases occurring in shipment ..... J. I. LAURITZEN, L. L. HARTER, and W. A. WHITNEY 411

Some viruses affecting spinach, and certain aspects of insect transmission ..... ISMÉ A. HOGGAN 446

Records of citrus scab mainly from herbarium specimens of the genus Citrus in England and the United States. ANNA E. JENKINS and H. S. FAWCETT 475

Phytopathological Notes 483

Physiologic specialization of Tilletia tritici on emmer. W. E. BRENTZEL

Germination of the oospores of Pseudoperonospora humuli. E. N. BRESSMAN and R. A. NICHOLS

The spermatia of flax rust, Melampsora lini ..... RUTH F. ALLEN

A cork-borer method for inoculating trees ..... ERNEST WRIGHT

Report of the twenty-fourth annual meeting of The American Phytopathological Society 489

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PHYTOPATHOLOGY

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ENVIRONMENTAL FACTORS IN RELATION TO SNAP-BEAN DISEASES OCCURRING IN SHIPMENT

J. I. Lauritzen, L. L. Harter, and W. A. Whitney

(Accepted for publication July 28, 1932)

INTRODUCTION

Anthracnose (*Colletotrichum lindemuthianum* (Sacc. and Magn.) Bri. and Cav.), bacterial blight (*Bacterium phaseoli* E.F.S. or *Bact. medicaginis* var. *phaseolicola* (Burk.) Link & Hall), watery soft rot (*Sclerotinia sclerotiorum* (Lib.) de Bary), cottony leak (*Pythium butleri* Subr.), soil rot (*Rhizoctonia solani* Kühn), southern blight (*Sclerotium rolfsii* Sacc.), Rhizopus soft rot (*R. tritici* Saito and *R. nigricans* Ehrnb.), and gray mold rot (*Botrytis cinerea* Pers.) are the principal transit diseases of snap beans, *Phaseolus vulgaris* L.

Some idea of the distribution and relative importance of some of these diseases may be had from the data presented in tables 1 and 2, obtained from market-inspection certificates, issued by the Division of Fruits and Vegetables, Bureau of Agricultural Economics, U. S. Department of Agriculture. The certificates employed represent all the car-lot inspections made at terminal markets in the United States during the years 1922 to 1928, inclusive, and deal with questions of grade and condition, including disease.

Table 1 indicates the States and geographical districts from which the cars originated. It does not, however, show the number of times that a particular disease originated from a particular State or district. It will be seen that watery soft rot is present in all the districts and in most, if not all, of the States. Anthracnose shows the next widest distribution and blight, the third. Some States, mostly along the South Atlantic and Gulf Coast, show the presence of all the diseases.

Table 2 shows that out of 3,107 cars inspected at all the terminal markets in the United States during the years 1922 to 1928, 15 per cent showed watery soft rot; 6.7 per cent, anthracnose; 3 per cent, bacterial blight; 2.3 per cent, soil rot; 1.3 per cent, Rhizopus soft rot; 0.8 per cent, gray mold rot; 3.1 per cent, other diseases (including slimy soft rot and powdery mildew, etc.); 17.1 per cent, injuries (including scarring, insect injury, freezing, etc.); and 50.6, showing no disease. It should be stated that the 15 per cent watery soft rot probably includes some cottony leak that was not distinguished from the former until 1925 (4).

TABLE 1.—*Distribution of the principal transit diseases of beans by States and geographical units*

Unit	State	Anthracnose	Blight	Watery soft rot	Soil rot	Rhizopus soft rot	Slimy soft rot	Gray mold rot
South Atlantic and Gulf Coast	Virginia	+[a]	+	+				+
	North Carolina	+	+	+				
	South Carolina	+	+	+	+	+	+	
	Georgia	+	+	+				+
	Florida	+	+	+	+	+	+	+
	Alabama	+		+		+		+
	Mississippi	+	+	+	+	+	+	+
	Louisiana	+	+	+	+	+	+	+
	Texas	+	+	+	+	+	+	+
Middle Atlantic	New York	+		+				
	New Jersey	+						
	Delaware	+						
	Maryland	+	+	+	+			
South Central East	Kentucky	+						
	Tennessee	+	+	+	+	+		+
South Central West	Oklahoma			+				
	Missouri			+				
	Arkansas	+		+				
North Central	Ohio	+		+	+			
	Michigan			+				
	Illinois			+				
West and Far West	Colorado		+					
	Oregon							
	California			+				

[a] (+) = Presence of disease.

Aside from the work of Ramsey (10) on Sclerotinia soft rot and Harter and Whitney's investigations (4) on cottony leak, the information available on the transit diseases of beans is limited largely to casual observations. Fulton (3) reports that anthracnose probably develops during the marketing of beans on lots that were apparently sound when picked.

Hedgcock (6) described Rhizoctonia lesions on bean pods. Fulton (3) reports damping off of bean seedlings, a dry rot of the stem, and lesions on the pods, all caused by Rhizoctonia. He believes that infection of pods is associated with injury of some kind. He states that the fungus often causes a bronzing in the absence of the development of lesions. Barrus (1) claims that Rhizoctonia attacks only pods lying in contact with the soil.

Ramsey (10), in his extensive studies on some of the species of Sclerotinia causing decay of vegetables, found that, although *S. sclerotiorum*, *S. intermedia* Ramsey, *S. minor* Jagger, and *S. ricini* Godfrey are able to

TABLE 2.—*Relative losses in beans in transit due to diseases and injuries*

Condition	Cars affected		Extremes of decay in cars
	Number	Per cent	Per cent
Watery soft rot	474[a]	15.0	T[d] to 33.0
Anthracnose	207	6.7	T to 38.5
Bacterial blight	93	3.0	T to 75.0
Soil rot	71	2.3	T to 62.0
Rhizopus soft rot	39	1.3	T to 80.0
Gray mold rot	25	0.8	T to 16.0
Other diseases[b]	95	3.1	
Injuries[c]	530	17.1	
No decay	1,573	50.6	

[a] Total number of cars inspected from 1922 to 1928, inclusive—3,107.

[b] Including slimy soft rot, powdery mildew, undetermined soft rot, an unnamed decay, and an unidentified nest.

[c] Including scarring, insect (principally corn-ear worm) injury, freezing, and other uncatalogued blemishes.

[d] T—Trace or less than 1 per cent.

infect and decay beans, *S. sclerotiorum* is responsible for most of the decay occurring in transit. On the losses occurring in transit, he has the following to say (p. 600): "After selecting at random 42 cars of green beans, which on inspection at market showed Sclerotinia decay, it was found that the percentage of infection ranged from 5 per cent in some cars to as high as 75 per cent in others, the average for all the cars being 24 per cent." He obtained infection of slices of turnip and carrots and of lettuce leaves by means of ascospores and of beans by means of mycelium and sclerotia of *S. sclerotiorum*. The range of temperature at which ascospores were germinated in sterile water and nutrient broth was 3–4° to 30–31°.[1] The fungus grew on potato-dextrose agar at temperatures extending from 0° to 32–33° and infected beans from 0° to 27–30°. Harter and Whitney (4) reported the common occurrence of cottony leak during the warmer months of the year in snap beans shipped to northern markets from the South and that the disease probably originated in the field. They compared the decay produced by *Pythium butleri* (*P. aphanidermatum*) with that produced by *S. sclerotiorum*, *Rhizoctonia solani*, *Botrytis cinerea*, *Rhizopus tritici*, and *R. nigricans*. Pythium caused a more rapid decay at warm temperatures (about 25°) than the other pathogens. They (5) found the maximum, minimum, and optimum temperature for growth of *P. butleri* on corn-meal agar to be approximately 38.8°, 6°, and 32°, respectively.

[1] Unless otherwise indicated in the text, the temperatures recorded throughout this paper are expressed in °C.

Important considerations in connection with the transit diseases of beans[2] are source of inoculum, origin of infection (whether in field or in transit), and the effect of temperature and time on infection and the subsequent development of the disease.

In an effort to throw additional light on some of these questions, experiments were conducted during the 6 years 1925 to 1931, at Arlington Experiment Farm, Rosslyn, Virginia.

MATERIALS AND METHODS

Pathogens. The pathogens used in these experiments and their origins are as follows: *Colletotrichum lindemuthianum*, bean; *Bacterium phaseoli*, bean and Lima bean; *Sclerotinia sclerotiorum*, sweet potato; *Pythium butleri*, sweet potato; *Rhizoctonia solani*, bean; *Sclerotium rolfsii*, bean; *Rhizopus tritici*, sweet potato; *R. nigricans*, sweet potato; and *Botrytis cinerea*, pepper. Some experiments were conducted with *C. lindemuthianum* in which the inoculum was obtained from anthracnose lesions on bean pods.

Host. The beans used were obtained on the Washington market and originated in Florida. They were purchased the morning they arrived and used at once in the experiments. The Bountiful variety was mostly used, but each of the varieties Black Valentine, Longfellow, Giant Stringless Green Pod, and an unidentified variety were used once. In all the experiments, defective, abnormal, or infected beans were sorted out.

Technique. The inoculations in case of all the pathogens, except *Colletotrichum lindemuthianum* and *Bacterium phaseoli*, were made in a similar manner. A bit of the fungous growth was placed in a small slit made in the pod, which was then placed in the center of a 4-qt. till basketful of beans. In some of the trials, involving infection by species of Rhizopus, dependence was placed upon these fungi normally present on the beans. In the experiments with anthracnose, spores were either inserted into pods with a needle or sprayed on them with an atomizer. Whenever inoculation experiments were attempted with the blight organism the inoculum was introduced into the bean pods by means of a needle puncture or the beans were atomized with a water suspension of the bacterium. In some of these experiments with the latter pathogen, beans known to be contaminated and possibly infected with blight in the field were used.

Equipment and Control of Temperature and Humidity. The beans were stored either in ventilated insulated chambers (8) or in galvanized iron chambers (9) the temperature of which was controlled by thermoregulators. The humidity of all chambers was governed by water in evaporation pans.

[2] Use of the term "bean" or "beans" throughout this paper signifies snap beans and not shelled beans.

EXPERIMENTAL WORK AND RESULTS

Anthracnose. The ability of *Colletotrichum lindemuthianum* to infect snap beans (Longfellow and Bountiful varieties) has been demonstrated by two types of experiment—one in which spores were inserted into the beans by needle pricks and the other in which the spores from culture or from infected beans were sprayed on the pods with an atomizer. The development of anthracnose lesions has been observed in inoculated sound beans and in inoculated and noninoculated beans contaminated and possibly infected in the field.

Pods inoculated by inserting spores with a needle became infected (Experiment 1, Table 3) within 2 days at temperatures of 15.5°, 19°, 22.5°, 28°, 31°, and 33°. These pods were placed in the center of 4-qt. till baskets of beans (Bountiful variety) and the baskets stored at the various temperatures. At the end of 3 days, infection was evident at 12°. A temperature of 33° is 2° higher than the maximum obtained by Edgerton (2) when the organism was grown on culture media. In a second experiment (Experiment 2, Table 3), infection occurred in 4 days at temperatures ranging from 8° to 31°. Checks were run in both experiments and negative results were obtained at all temperatures.

A quantity of beans (Bountiful variety) was purchased, showing the presence of a slight amount of anthracnose. All beans bearing anthracnose lesions, were carefully culled out and the apparently sound ones were divided into 3 lots. One lot was placed in 4-qt. till baskets and, without further treatment, was stored at various temperatures. The second lot was sprayed with *Colletotrichum lindemuthianum* spores from test-tube cultures grown on bean pods. The third lot was inoculated by spraying with a water suspension of spores obtained from anthracnose lesions on living pods. After inoculation the last 2 lots were transferred to 4-qt. till baskets and stored at the various temperatures with the noninoculated beans (checks).

The results obtained from the checks (Table 4) illustrate the development of anthracnose lesions at various temperatures in beans free from any visible signs of infection at the time they were stored. Lesions developed over a range of temperature from 7.5° to 29°. Lesions had already developed in 6 days at temperatures of 15°, 17.5°, 21°, 25°, and 29°. At temperatures of 12.5°, 10°, and 7.5°, the first lesions were observed after 8, 11, and 18 days, respectively. The lesions may have resulted from incipient infections or new infections or from both.

That lesions may result from the inoculation of beans with spores of anthracnose is evident from the results (Table 4) obtained from the beans inoculated with spores from culture and from live pods, as compared with those obtained from the checks. In most instances the inoculated beans

TABLE 3.—*Relation of temperature to infection of beans (Bountiful variety) inoculated with Colletotrichum lindemuthianum (needle-prick inoculation)*

Experiment 1								Experiment 2				
			Infection after different periods of time in days								Infection in 4 days	
			2		3		7					
Temperature	Relative humidity	Number of inoculations	Infection	Diameter of lesions	Infection	Diameter of lesions	Infection	Temperature	Relative humidity	Number of inoculations	Infection	Diameter of lesions
°C.	*Per ct*		*Per ct*	*Mm.*	*Per ct*	*Mm.*	*Per ct*	°C.	*Per ct*		*Per ct*	*Mm.*
33.0	84	12	100	2.5				35.5	93	30	0[a]	
31.0	92	17	100	2.5				31.0	95	30	60	2.5
28.0	83	14	100	4.0				28.0	80	30	87	3.0
22.5	88	11	91	2.5				23.0	92	30	87	3.0
19.0	89	12	100	2.5				17.5	89	30	93	3.0
15.5	93	11	100	1.0	100	3.5		15.5	94	30	90	2.0
12.0	92	14	0		100	Just started		12.0	95	30	100	2.75
8.0	98	12	0				0	8.0	93	30	53	2.0
6.0	97	11	0				0					

[a] There were lesions in 30 per cent of the punctures at this temperature, but they were abnormal. There was a reddening of all the punctures at this temperature.

TABLE 4.—*Influence of temperature on the development of anthracnose lesions in field-contaminated[a] beans (Bountiful variety), noninoculated (artificially), and inoculated with a water spore suspension of Colletotrichum lindemuthianum*

Temperature	Relative humidity	Percentage of infection after different periods of time in days																	
		Inoculated with spores from culture						Inoculated with spores from lesions on bean pods						Noninoculated beans					
		6	8	11	14	16	18	6	8	11	14	16	18	6	8	11	14	16	18
°C.	*Per ct.*																		
34.0	91	0.0						0.0						0.0					
29.0	96	0.0						0.0						2.6					
28.0	94	10.0						0.0						0.0					
25.0	93	49.1						65.8						20.0					
21.0	92	34.6						55.6						26.3					
17.5	91	9.8	40.2	67.0				7.0	40.0	94.8				8.3	20.9	35.9	37.2		
15.0	93	7.4	32.0	68.9				3.4	22.3	80.5				6.5	21.6	31.7	36.0		
12.5	94	0.0	0.0	16.2	56.9			0.0	1.8	14.3	67.1			0.0	1.9	8.8	14.0		
10.0	98	0.0	0.0	1.8	12.7			0.0	0.0	0.0	16.7			0.0	0.0	0.7	5.2		
7.5	91	0.0	0.0	0.0	1.8	3.4	7.8	0.0	0.0	0.0	0.9	2.7	6.2	0.0	0.0	0.0	0.0	0.0	0.7
2.0	97	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0		0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
0.9	95	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0		0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

[a] A few of the pods showed anthracnose lesions when purchased. These pods were carefully sorted out before starting the experiment.

gave a marked increase in infection over the checks, especially if time enough was allowed for infection resulting from inoculation to take place at any particular temperature under consideration. On the whole, the percentage of infection was higher in beans inoculated with spores taken from the living bean pods than in those inoculated with spores from culture.

Two experiments were run in which beans (Bountiful variety), free or nearly free from field contamination or infection, were inoculated by spraying with a spore suspension of *Colletotrichum lindemuthianum*. One lesion developed at one temperature (31°) in the checks in one of the experiments; the checks in the second experiment showed no evidence of anthracnose. In the first experiment, one lot of beans was inoculated with spores from culture and another lot with spores from the host. In the second experiment spores from the host alone were used. One 4-qt. till basket of inoculated and one of noninoculated beans were stored at different temperatures from 2° to 31° (Table 5). Inspections were made in the

TABLE 5.—*Relation of temperature and time to infection of sound beans (Bountiful variety) inoculated with a spore suspension of Colletotrichum lindemuthianum*[a]

Temperature	Relative humidity	Percentage of beans infected after different periods of time in days													
		3	4	5	6	7	8	9	10	11	12	13	14	15	16
°C.	*Per ct.*														
31	93	0	.....	0	.....	0	.....	0							
29	97	0	0	0	0	0	0	0							
27	92	.....	0	4	40	.....	76	.....							
25	95	0	0	28	88	91	100	98							
22	93	0	0	8	64	88	99	98							
17.5	89	0	0	0	0	18	17	72	70	89		96			
15.5	92	0	0	0	0	1	0	23	40	73	76	94	86	97	96
12	93	0	0	0	0	0	0	0.8	0	0	36	25	68	71	84
10	98	0	0	0	0	0	0	0	0	0	2	0	20	20	67
7	92	0	0	0	0	0	0	0	0	0	0	0	0.9	0	0.9
5.5	95	0	0	0	0	0	0	0	0	0	.....	0	.....	0	.....
2	97	0	0	0	0	0	0	0	0	0	0	.....	0	.....	0

[a] The data taken after 3, 5, 7, 9, 12, and 14 days were obtained from one experiment and those for 4, 6, 8, 10, 11, 13, 15, and 16, from a second experiment.

first experiment after 3, 5, 7, 9, 12, 14, and 16 days and in the second after 4, 5, 6, 8, 10, 11, 13, and 15 days. Table 5 contains the composite results of these experiments with snap beans inoculated with a spore suspension from the host, and table 6 shows the results from the one experiment in which beans were inoculated with spores from culture and host. The results given in table 5 are represented graphically in figure 1.

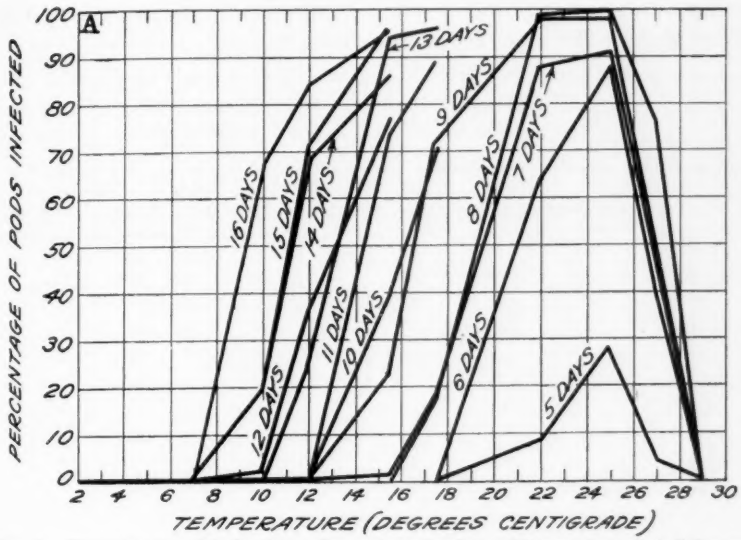

FIG. 1. Percentage of infection of one 4-qt. basket of beans inoculated by spraying with a water spore suspension of *Colletotrichum lindemuthianum* from living bean pods, after different periods of time at various temperatures.

The earliest infection was observed in 5 days. In the first experiment an inspection was not made on the fourth day. However, judged by the amount of infection occurring in 5 days in this particular experiment, there probably was no infection in 4 days, either when inoculated with spores from pure culture or with spores from the host (Table 6). If all the results are taken into consideration, the earliest infections were limited to temperatures of 22°, 25°, and 27°. As the temperature fell below 22°, the earliest infection occurred in 7 days at 15.5° and 17.5°, in 9 days at 12° and 12.5°, in 12 days at 10°, and in 14 days at 7°. No infection occurred at 2° or 5.5° in 16 days. The highest temperature at which infection occurred was 27°, which approximates the maximum (27.2°) (7) obtained for infection of growing bean plants. The lowest temperature at which infection was obtained is 7°, about 7° below the lowest temperature (14.4°) at which infection of growing bean plants was obtained (7). In the latter case the plants were exposed to the various temperatures at humidities favorable to infection for 24 hours, when they were removed to humidities that did not permit infection, whereas, in the former case, the bean pods were stored at favorable humidities at temperatures of 15.5° and below for periods of 11 to 18 days. This difference in time of exposure to conditions favorable to infection, as well as the physiological differences between plants and pods, may account for the difference in the lower temperature limits of infection in the two cases. The optimum temperature

TABLE 6.—*Influence of kind of inoculum on infection of beans (Bountiful variety) by Colletotrichum lindemuthianum at different temperatures*

Temperature	Relative humidity	Percentage of infection after different periods of time in days													
		3		5		7		9		12		14		16	
		Culture[a]	Host	Culture	Host	Culture	Host	Culture	Host	Culture	Host	Culture	Host	Culture	Host
° C.	*Per ct.*														
31	93	0	0	0	0	0	0	0	0						
29	97	0	0	0	0	0	0	0	0						
25	94	0	0	0.9	7	32	91	43	98						
22	93	0	0	0.8	3	58	88	69	98						
17.5	91	0	0	0	0	8	18	22	72						
15.5	90	0	0	0	0	0	1	10	23	42	76	43	86	50	96
12.5	93	0	0	0	0	0	0	0	0.8	20	36	58	68	70	83
10	98	0	0	0	0	0	0	0	0	0.8	2	13	67	65	67
7	91	0	0	0	0	0	0	0	0	0	0	0	0.9	0	0.9
2	97	0	0	0	0	0	0	0	0	0	0	0	0	0	0

[a] In the one instance the beans were inoculated with a spore suspension from pure culture on sterile bean pods and in the other with a spore suspension from lesions on live beans.

for infection of the pods ranges from 22° to 25°, the highest percentage of infection usually occurring at 25°. The optimum temperature for growth of Colletotrichum on culture media was 21° to 23° (2).

It will be noted (Table 6) that the percentage of infection at any given temperature and period of time was higher in beans inoculated with spores from the living pods than from the culture. Infection was also obtained at a lower temperature (7° as compared with 10°) in the former than in the latter case. This fact, taken in connection with similar results given in table 4, suggests that spores taken directly from the host may be more pathogenic than spores from artificial culture media, or there may be a loss of virulence in culture. It may, however, merely mean that a heavier spore suspension was used in the one case than in the other.

Bacterial Blight. *Bacterium phaseoli* and *Bact. medicaginis* var. *phaseolicola* cause lesions so similar on bean pods that they are not easily distinguishable, and no attempt has been made in these investigations to differentiate between lesions produced by them. *Bacterium phaseoli* alone has been used in the inoculation experiments.

The ability of *Bacterium phaseoli* to infect snap-bean pods detached from vines was tested in two ways: through wounds and by spraying with a water suspension of the pathogen.

Two experiments were conducted in which the needle-prick method was used, and infection (Table 7) took place readily over a range of tempera-

TABLE 7.—*Relation of temperature to infection of beans*[a] *by Bacterium phaseoli (needle punctures)*

Experiment 1													Experiment 2									
			Infection and decay after different periods of time in days											Infection and decay after different periods of time in days								
			4		6		10		15					4		6		8		11		
Temperature	Relative humidity	Number of inoculations	Infection		Diameter of lesions		Infection		Diameter of lesions		Infection		Diameter of lesions	Infection		Diameter of lesions	Infection		Diameter of lesions	Infection		Diameter of lesions
°C.	Per cent		Per cent		Mm.		Per cent		Mm.		Per cent		Mm.	Per cent		Mm.	Per cent		Mm.	Per cent		Mm.
35.5	95	15	0											35.5	93	11	0					
31	91	15	93		4									31	95	28	64		25			
24	95	14	79		2.5									24.5	94	33	88		2.25			
21.5	91	15	0		0		66		3		100		4.5	21	91	30	7		–			
15.5	93	15	0		0		0		0		27		3	15.5	94	32	0		0		25		3.25			
12	90	15	0		0		0		0		0		0	12	95	31	0		0		0		0	0		0
8	93	15	0		0		0		0		0		7	–	8	93	30	0		0		0		0	0		10	4.5
6	95	15	0		0		0		0		0		0	0	2	98	32	0		0		0		0	0		56	1.25
2	97	15	0		0		0		0		27		1									

[a] The Black Valentine variety was used in Experiment 1; the Bountiful variety in Experiment 2.

[b] Pods partially decayed by Botrytis.

ture extending from 2° to 31°. No infection occurred at 35.5°. Considerable infection had occurred at temperatures of 24°, 24.5°, and 31° in 4 days in the two experiments, and in one of the experiments 7 per cent of the inoculations at 21° resulted in infection in 4 days. In one experiment at 15.5° there was 25 per cent infection in 6 days and, in the other, 27 per cent in 10 days. In one experiment there was no infection in 8 days at 12° and none in 15 days in the other. In the latter case the results were complicated by infection by *Botrytis cinerea*. Infection was evident in 11 days at 8° and 2° in one experiment and in the other experiment, at 2°, but not at 6° in 15 days.

Three experiments were conducted in which a water suspension of *Bacterium phaseoli* was sprayed on snap-bean pods by means of an atomizer. In the first 2 experiments, blight was present on the beans when purchased. In the third it was absent. In the former two all the infected pods were sorted out before inoculating the remaining beans. In each case the beans were stored at various temperatures in 4-qt. till baskets. The temperature range employed for each of the 3 experiments was as follows: 5.5° to 29° (11 temperatures), 2° to 31° (10 temperatures), and 1.2° to 29° (11 temperatures), respectively. The relative humidities were above 90 per cent in all cases. All 3 experiments yielded negative results so far as the effects of inoculation are concerned. This fact is not so clear in those experiments in which blight infection was present in the original stock as it is in the one in which blight was absent. The results of one of these experiments in which blight was originally present are given in table 8. The findings of the second experiment were similar. There was even slightly less infection in the inoculated as compared with noninoculated beans (Table 8), as indicated by the compensated percentages that represent the difference between the percentage of infection in the checks and the inoculated beans. The absence of infection was not due to a lack of virulence on the part of the pathogen because infection resulted in beans inoculated by puncturing the pods with a needle contaminated with the organism and stored at the various temperatures along with the sprayed beans. Seventy-two per cent of the 25 needle-prick inoculations at 28° resulted in infection in 3 days, 56 per cent at 21° in 11 days, 8 per cent at 17° in 11 days, and 24 per cent at 15° in 11 days. No checks were run in connection with the other 2 experiments, but the needle-prick method of inoculation has always yielded positive results in our experiments.

The results obtained from inoculations with a bacterial suspension may be interpreted in one of two ways: either the conditions suitable for infection were not met or *Bacterium phaseoli* normally lacks the ability to infect uninjured pods detached from the vines.

TABLE 8.—*Development of blight at different temperatures in beans (Bountiful variety) inoculated with a water suspension of Bacterium phaseoli*

Temperature	Relative humidity	Beans used	Inoculated beans: Percentage of beans infected after different periods of time in days						Beans used	Noninoculated beans (checks): Percentage of beans infected after different periods of time in days						Compensated percentages: Differences in percentage of infection in inoculated and noninoculated beans after different periods of time in days					
			6	8	11	14	16	18		6	8	11	14	16	18	6	8	11	14	16	18
°C.	*Per ct.*	*Number*							*Number*												
29	94	141	14.2						90	6.7						+ 7.5					
28	96	121	3.3						152	6.6						− 3.3					
25	93	146	14.4						99	16.2						− 1.8					
21	92	152	7.2						147	12.9						− 5.7					
17	91	138	4.4	37.0	32.0	38.4			148	5.4	22.3	49.3	53.2			− 1.0	+ 14.7	− 17.3	− 14.8		
15	93	144	4.9	20.8	29.2	41			139	5.8	15.2	40.3	43.9			− 0.9	+ 5.6	− 11.1	− 2.9		
12	94	117		10.3	19.7	24.8			159		9.4	14.6	26.4				+ 0.9	+ 5.1	− 1.6		
10	98	136			3.7	11			135			5.9	10.4					− 2.2	+ 0.6		
7	91	121				5	11.6	12.4	135				10.4	15.6	13.3				− 5.4	− 4.0	− 0.9
2	97	116					12.9	23.3	145					18.6	33.8					− 5.7	− 10.5
1.2	95	112					16.1	36.6	150					22	44.7					− 5.9	− 8.1

The results submitted in table 8, as well as those obtained from the other experiment in which blight was found present in the original stock, indicate that blight may develop in snap beans stored at various temperatures in which there were no visible signs of the disease at the beginning of storage. Blight lesions developed at temperatures from 1.2° to 29°. After 6 days' storage infection in noninoculated beans varied from 5.4 to 16.2 per cent at temperatures from 15° to 29°. There was an increase in the percentage of infection with the lapse of time at temperatures below 21°.

In order to give a more adequate picture of the development of blight as related to temperature and time, daily readings were taken of the blight present in beans of the Bountiful and Giant Stringless Green Pod varieties stored at 11 different temperatures in 4-qt. till baskets. About 7 per cent of the pods of both varieties were infected when purchased on the market. They had been 3 days *en route* from Canal Point, Florida, to Washington, D. C. The blighted pods were carefully sorted out and the rest of the beans thoroughly mixed to insure a fairly uniform distribution of contaminated beans or those that may have been carrying incipient, invisible infections.

The results obtained from the 2 varieties were so similar that only those of the Giant Stringless Green Pod will be presented (Table 9). Blight developed at all temperatures, although very little at 35°, indicating that this temperature is approaching the maximum. There is a wide range (10°

TABLE 9.—*Influence of temperature and time on the development of blight in beans of the variety Giant Stringless Green Pod*

Temperature	Relative humidity	Pods used	Percentage of beans infected after different periods of time in days								
			1	2	3	4	5	6	7	8	11
° C.	*Per cent*	*Number*									
35.0	92	288	0.3	0.3	0.3	0.3	0.3	0.3	0.3		
31.0	92	224	0.9	1.4	2.2	2.7	4.0	6.7	8.9		
28.5	95	260	1.2	3.5	5.4	8.1	8.1	9.5	8.8		
24.5	91	233	4.3	6.8	9.4	9.0	11.2	13.3	12.9	14.2	
24.5	91	249	2.0	5.2	6.4	6.4	8.0	8.0	11.2	15.3	
19.5	94	298	5.0	7.1	6.7	8.4	8.4	9.1	9.1	13.1	
19.5	94	274	6.2	7.7	9.1	11.0	10.2	13.1	11.7	11.3	
17.5	91	244	4.9	4.1	5.7	9.0	8.6	11.1	11.9	12.3	
15.0	94	286	3.2	4.5	5.2	8.4	11.2	12.6	12.6	12.6	
13.0	93	243	1.7	3.3	4.9	4.9	8.2	9.1	8.2	9.1	
10.0	99	268	1.9	3.0	3.7	4.9	9.3	9.3	11.6	11.9	
7.0	93	234	1.3	2.6	2.6	2.1	2.6	5.1	5.6	6.4	7.2
2.0	97	263	.....	2.5	2.5	2.7	4.6	4.2	4.2	4.2	4.2

to 24.5°) of temperature at which there was little difference in the percentage of pods infected. There is some irregularity in the percentage of infection within this range, persisting at certain temperatures throughout the duration of storage (compare results at 10°, 13°, and 15°). As the temperature fell below 10° the percentage of infection generally decreased. In the case of the variety Bountiful there was a gradual decrease as the temperature fell below 17.5°.

New infections may not normally occur in pods detached from the vines, but blight will develop over a wide range of temperature in contaminated lots sorted free of any visible signs of infection.

Watery Soft Rot. Infection and the spread of watery soft rot (*Sclerotinia sclerotiorum*) during transit are probably primarily effected by means of mycelium and possibly in some instances by ascospores and sclerotia. Its development probably results from the inclusion of infected beans and possibly of ascospores and sclerotia in the shipping package. The mycelium radiates from local points of infection and soon forms a continuous mass of decayed beans and mycelium, known in the trade as a "nest."

The rate of development of these nests, at various temperatures, is an important element in determining the losses occasioned during transit.

A series of 5 experiments was conducted from 1926 to 1928, with 3 varieties of beans (Bountiful in 3 experiments, Longfellow in 1, and an unidentified variety in 1. The results of 2 experiments are presented in tabulated form in table 10. The comparative rates of decay in the 5 experiments at various temperatures after a given period of time are represented graphically in figure 2, A. Infection was obtained within a temperature range of 0° to 28°. Infection occurred in 1 experiment at 27.5° in 5 days (Table 10) and in a second at 28° in 7 days. No infection developed in a third in 4 days at 28° or in a fourth in 5 days at 28.5°. A temperature of 31° was employed in 1 experiment and a temperature of 32° in 2 experiments. There was no infection in the former in 4 days nor in the latter in 5 and 7 days, respectively. These temperature limits for infection of snap beans correspond almost exactly with those (0° to 27–28°) obtained by Ramsey (10).

Infection occurs very slowly at 0° to 2°. In 2 experiments in which the duration of storage was 20 days at 2°, the number of pods infected besides the inoculated bean was 1 and 2, respectively. In 2 experiments infection was limited to the inoculated beans after 15 days' storage at 1.8°. In a third experiment 1 pod, besides the inoculated bean, became infected in 15 days at 1.8°. Infection was not observed in a shorter time at this temperature. Infection occurred in 9 days at 5.5° to 6°, in 7 days at 8° (data not given in table), and in 4 days at 12° (Table 10). The maximum number of infections occurred at temperatures ranging from 19° to 24.3° in 4 of

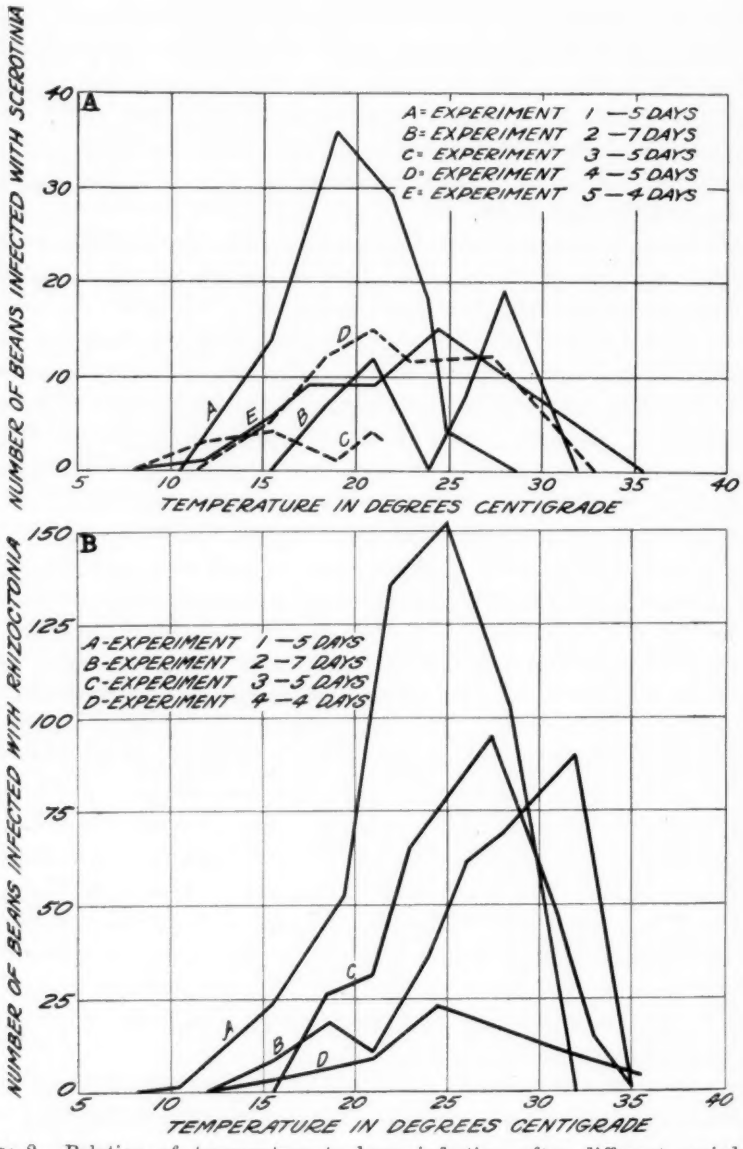
A
A= EXPERIMENT 1 —5 DAYS
B= EXPERIMENT 2 —7 DAYS
C= EXPERIMENT 3 —5 DAYS
D= EXPERIMENT 4 —5 DAYS
E= EXPERIMENT 5 —4 DAYS
NUMBER OF BEANS INFECTED WITH SCEROTINIA
TEMPERATURE IN DEGREES CENTIGRADE
B
A-EXPERIMENT 1 —5 DAYS
B-EXPERIMENT 2 —7 DAYS
C-EXPERIMENT 3 —5 DAYS
D-EXPERIMENT 4 —4 DAYS
NUMBER OF BEANS INFECTED WITH RHIZOCTONIA
TEMPERATURE IN DEGREES CENTIGRADE

FIG. 2. Relation of temperature to bean infection after different periods. A. *Sclerotinia sclerotiorum*. B. *Rhizoctonia solani*.

the 5 experiments. In the fifth experiment the largest number of infections occurred at 28° (Fig. 2, A). It is believed that this temperature is above the optimum, thought to lie between 19° and 24°. This fact is indicated from the average optimum obtained from the 3 experiments in which

TABLE 10.—*Relation of temperature and time to infection of snap-beans[a] by Sclerotina sclerotiorum*

		Experiment 1 Infection and decay after different periods of time in days								Experiment 2 Infection and decay after different periods of time in days											
		5		11		15		16				4		6		8		11		15	
Temperature	Relative humidity	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Temperature	Relative humidity	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest
° *C.*	*Per cent*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	° *C.*	*Per cent*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*
35.2	89	0	0							35.5	93	0	0								
33	84	0	0							24.5	94	15	46								
27.5	83	12	65							21	91	9	41								
23	88	12	82							17.5	89	9	34								
21	90	15	82							15.5	94	6	28	21	48						
18.5	89	12	70							12	95	1	.....	6	35						
15.5	93	5	32	71	122	33	82	163	140	8	93	0	0	5	23	2	20	6	33		
11.5	92	0	0	27	50	125	125	125	140	2	98	0	0	0	0	0	0	1[b]	.....	1[b]	.....
7.5	98	0	.....	3	27	41	82	50	100	0	97	0	0	0	0	.....	.....	0	0	1[b]	.....
6	98	0	.....	1	20	14	52	10	50												
2	97	0	0	0	0	1	18	1[b]	.....												

[a] An unidentified variety was used in Experiment 1 and the Longfellow variety in Experiment 2.
[b] Only the inoculated bean was infected.

inspection was made in 5 days (Fig. 2, A). The lack of uniformity in the results of the different experiments at a given temperature is explained, in part, by the fact that the results of individual inoculations are expected to vary in the amount of decay they produce.

Cottony Leak. The nesting of beans, caused by *Pythium butleri* (*aphanidermatum*), is very similar (4) to that produced by *Sclerotinia sclerotiorum* and, before 1927, the decays produced by these organisms were not differentiated. Although it is not definitely known just how decay is initiated in transit, it is believed to be due largely to the inclusion of infected pods in the hampers of beans at the time of picking. Healthy beans become infected if placed in contact with diseased pods. As a result, nests, sometimes involving several quarts of beans, are formed in the containers during the shipping period.

In order to study infection and the development of cottony leak at various temperatures, 3 tests (only 1 reported in tabulated form, Table 11) were made in which the beans (Bountiful, Longfellow, and an unidentified variety) were inoculated and stored in the same fashion as in the experiments with watery soft rot. Infection occurred at 12° to 35.6°. Higher temperatures than 35.6° were not employed. Infection was limited to the inoculated bean at 12° and 15.5°, even after 16 days' storage. This temperature is 6° above that of the minimum growth of the pathogen in pure culture, as obtained by Harter and Whitney (5). The maximum amount of decay was obtained at 31°, above and below which decay decreased as the temperature was raised or lowered. The shortest time required for infection to occur at the higher temperatures was not determined. Judging by the amount present in 2 days at temperatures from 21° to 35°, its development is very rapid.

Soil Rot. Field infection of bean pods probably originates in most, if not all, instances from contact of the pods with the soil, although it is conceivable that it may occur in other ways, such as from the splashing of infested soil onto the pods or vines during heavy rains. Infection of beans in transit by *Rhizoctonia solani* may result in nesting. As a rule, however, the fungus grows superficially through a lot of beans, infecting pods at local points and forming definite concentric lesions on the pods.

A series of 5 experiments was conducted from 1926–1928 in which 3 varieties of beans (Bountiful in 3 experiments, Black Valentine and an unidentified variety in each of the other 2) were used. The beans were inoculated and stored at various temperatures in the same manner as in the experiments with watery soft rot. The results of 3 experiments are given in table 12. The highest and lowest temperatures at which infection was obtained were 35.5° and 0.9°, respectively. Only in 1 experiment was infection observed at temperatures below 11.5°. The first evidence of in-

TABLE 11.—*Relation of temperature and time to infection of beans (unidentified variety) by Pythium butleri*

Temperature	Relative humidity	Infection and decay after different periods of time in days									
		2		3		5		11		16	
		Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected
°C.	*Per cent*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*
35.0	89	35	62	50	118						
33.0	84	31	53	48	136						
31.0	92	34	62	55	150						
28.0	83	26	26	35	60						
22.0	88	19	17	28	36						
21.0	90	8	5	7	4						
18.5	89	0	0	7	4						
15.5	93	0	0	0	0	–	1	5	1	–	1
12.0	92	0	0	0	0	0	0	–	1	–	1
7.5	98	0	0	0	0	0	0	0	0	0	0
5.5	98	0	0	0	0	0	0	0	0	0	0
2.0	97	0	0	0	0	0	0	0	0	0	0

TABLE 12.—*Relation of time and temperature to infection of beans[a] by Rhizoctonia solani*

Experiment 1					Experiment 2					Experiment 3						
Temperature	Relative humidity	Beans infected after indicated time (days)			Temperature	Relative humidity	Beans infected after indicated time (days)			Temperature	Relative humidity	Beans infected after indicated time (days)				
		5	16	20			5	11	16			5	6	8	11	15
° *C.*	*Per cent*	*Number*	*Number*	*Number*	° *C.*	*Per cent*	*Number*	*Number*	*Number*	° *C.*	*Per cent*	*Number*	*Number*	*Number*	*Number*	*Number*
32.0	92	0			35.5	89	1			35.5	93	4				
28.5	88	103			33.0	84	15			31.0	95	11				
25.0	97	152			31.0	92	46			24.5	94	23				
22.0	91	135			27.5	83	95			21.0	91	9				
19.0	90	53			23.0	88	65			15.5	94	3	1			
15.5	88	24			21.0	90	32			12.0	95	0	0	0	0	
10.5	93	2	3		18.5	89	27			8.0	93	0	0	0	0	0
8.0	98	0	1[b]		15.5	93	0	0	273	6.0	98	0	0	0	0	0
5.5	98	0	1[b]		11.5	92	0	0	4	2.0	97	0	0	0	0	0
2.0	97	0	0	1[b]	7.5	98	0	0	0							
0.9	97	0	0	1[b]	6.0	98	0	0	0							
					2.0	96	0	0	0							

[a] The variety Bountiful was used in Experiments 1 and 2 and an unidentified variety in Experiment 3. The number of infections at each temperature and period of time resulted from a single inoculation.

[b] Infection confined to inoculated pod.

fection in the inoculated pod at temperatures of 0.9° and 2° occurred in 20 days, and in 16 days at 5.5° and 8° (Experiment no. 1, Table 12). A temperature of 35° to 35.5° was employed in 4 out of the 5 experiments. Infection was obtained at this temperature in only 2 experiments. In the fifth experiment there was infection at 32°, the highest temperature employed. It would seem from these results that a temperature of 35° is near the maximum for infection. There was some variation in the temperature at which the maximum amount of infection (Table 12 and Fig. 2, B) occurred. The number of inoculations at a given temperature was here also a limiting factor. Furthermore, it is believed that a normal variation in a temperature optimum is to be expected. The optima obtained in all 5 experiments lie within the limits of 24° and 32°, respectively. Considerable infection may take place at 15.5° to 33°. Although infection has occurred in 5 days at 10.5° (Experiment 1, Table 12), it is usually very limited below 15.5°, even after 15 to 20 days' storage. The rate of development of the number of beans showing Rhizoctonia lesions between 20° and 30° is high. (Fig. 2, B.)

Southern Blight. Infection of beans by *Sclerotium rolfsii* during transit probably results from the inclusion in the pack of soil-contaminated bean pods in much the same way as in the case of *Sclerotina sclerotiorum* and *Rhizoctonia solani.* Nesting occurs in the same manner as it does in watery soft rot and cottony leak.

Three experiments (only 1 reported in tabulated form, Table 13) similar to those of watery soft rot were conducted with *Sclerotium rolfsii.* Infections were obtained at temperatures from 8° to 35.5° (Table 13). Judging by the number of pods (7 in 1 and 3 in another experiment) infected in 3 days at 35.6° in 11 days at 8° infection was limited to the mere beginnings of decay of the inoculated pod. No infection occurred at 6° in the 3 trials. The maximum number of infections in each trial in 3 and 4 days occurred at 31° (Fig. 3). As the temperature rose above or fell below 31°, the rate of decay declined rapidly. Infection had occurred at temperatures of 17.5°, 23°, 28°, 31°, and 35.5° in 2 days. Decay had just started at 17.5°. The earliest infection (inoculated pod) at 12° occurred in 5 days (data not included in table) and at 8° in 11 days (Table 13).

Rhizopus Soft Rot. Although no accurate survey has been made of the species of Rhizopus responsible for decay of beans in transit, *R. tritici* and *R. nigricans* are the only ones that have been noted. The species are sufficiently omnipresent to be a source of decay in beans in transit whenever the conditions are favorable for infection. Nesting is a characteristic of Rhizopus decay, although it is not so pronounced as in watery soft rot, cottony leak, and southern blight.

TABLE 13.—*Relation of temperature and time to infection and decay of bean pods (Longfellow variety) by Sclerotium rolfsii*

Temperature	Relative humidity	Infection and decay after different periods of time in days											
		2		3		6		8		11		15	
		Diameter of nest	Beans[a] infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected
° C.	*Per cent*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*
35.5	93	40	10	33	7								
31.0	95	38	11	90	53								
28.0	80	48	10	78	23								
23.0	92	28	4	63	25								
17.5	89	—	1	8	2	53	15						
12.0	95	0	0	0	0	–	1	–	1	9	1		
8.0	93	0	0	0	0	0	–	0	0	–	1		
6.0	98	0	0	0	0	0	0	0	0	0	0	0	0

[a] The beans infected at each temperature and after each period of time resulted from a single inoculation at the center of a till basketful of beans.

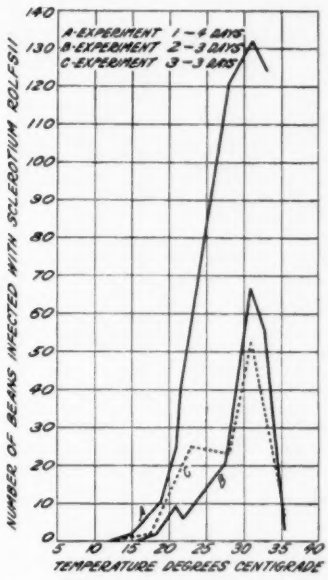

FIG. 3. Relation of temperature to the rate of decay of beans by *Sclerotium rolfsii* at different temperatures in 3 or 4 days in three experiments.

Two experiments were run with each of *Rhizopus tritici* and *R. nigricans* in which the beans were inoculated in the same manner as in the case of watery soft rot and stored at the various temperatures in 4-qt. till baskets. The diameter of the nest and the number of beans infected about a point of inoculation were used as criteria of decay. Data also were taken in connection with noninoculated beans (checks) stored at the various temperatures in the experiments dealing with the other diseases. The results of one of the experiments with inoculated beans are given in table 14 and those of the noninoculated beans in table 15.

There was little difference in the amount of infection or decay between bean pods inoculated with *Rhizopus tritici* and *R. nigricans* in either experiment. At 12° and 15.5°, inoculation with *R. tritici* resulted in infection in 1 experiment, while inoculation with *R. nigricans* caused decay in both. Inoculation of beans by either species resulted in heavier and more uniform infection than is the rule in beans stored without inoculation. The inclusion of an infected bean in the container at the time of shipment, therefore, would be expected to aggravate greatly the losses incurred during transit at temperatures favorable to infection and decay.

Infection has been obtained in noninoculated and inoculated beans over a range of 12° to 35.5°. No infection ever has been obtained at temperature below 12°, even after 16 to 20 days' storage, whether the beans were

TABLE 14.—*Relation of temperature to infection and decay of beans (Bountiful variety) by Rhizopus tritici and R. nigricans*

Temperature	Relative humidity	*Rhizopus tritici*						*Rhizopus nigricans*					
		Infection and decay after different periods of time in days											
		4		5		7	9	4		5		7	9
		Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Beans infected	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Beans infected
°C.	*Per cent*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Number*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Number*
33.0	84	43	100					29	120				
31.0	92	40	110					22	65				
28.0	83	8	90					14	100				
21.5	88	0	0	3	35			1	25				
19.0	89	0	0	1	—			0	0	3	45		
15.5	93	0	0	1	—			0	0	1	—		
12.0	92	0	0	1	10			0	0	1	—		
8.0	98	0	0	0	0	0	0	0	0	0	0	0	0
6.0	98	0	0	0	0	0	0	0	0	0	0	0	0
2.0	97	0	0	0	0	0	0	0	0	0	0	0	0

TABLE 15.—*Normal infection by species of Rhizopus in 5 lots of beans*[a]

Lot 1			Lot 2			Lot 3			Lot 4			Lot 5		
Temperature	Beans infected by Rhizopus	Storage period	Temperature	Beans infected by Rhizopus	Storage period	Temperature	Beans infected by Rhizopus	Storage period	Temperature	Beans infected by Rhizopus	Storage period	Temperature	Beans infected by Rhizopus	Storage period
°C.	*Number*	*Days*	°C.	*Number*	*Days*	°C.	*Number*	*Days*	°C.	*Number*	*Days*	°C.	*Number*	*Days*
35.5	3	4	35.5	9	4	33.0	4	4	35.2	Few	5	32.0	Many	5
31.0	0	4	31.0	0	4	31.0	8	4	33.0	1	5	29.0	5	5
28.0	3	4	28.0	0	4	28.0	0	4	31.0	3	5	25.0	5	5
24.5	6	4	24.5	0	4	21.5	0	5	28.0	0	5	23.5	0	5
23.0	2	4	23.0	0	4	19.0	0	5	23.0	0	5	22.0	0	5
21.0	0	6	21.0	0	6	15.5	0	5	21.0	1	5	19.0	0	5
17.5	8	6	17.5	0	6	12.0	0	5	18.5	0	5	15.5	0	5
15.5	3	6	15.5	1	6	8.0	0	7	15.5	Scattered infection	17	10.5	0	16
12.0	4	11	12.0	1	11	6.0	0	7	12.0	"	17	8.0	0	16
8.0	0	11	8.0	0	11	2.0	0	7	8.0	0	17	5.5	0	16
6.0	0	15	6.0	0	15				6.0	0	17	2.0	0	20
2.0	0	15	2.0	0	15				2.0	0	17			
0.0	0	15	0.0	0	15									

[a] The varieties of beans used were as follows: Lot 1, Longfellow; Lots 2, 3 and 5, Bountiful; Lot 4, an unidentified variety.

inoculated or not. Although the normal occurrence of Rhizopus infection at the various temperatures is somewhat erratic, it occurs commonly at temperature above 25° and sometimes in large amounts, especially if the temperature goes above 30°.

Gray mold rot. *Botrytis cinerea* is more or less omnipresent, and infection of beans may occur in transit whenever conditions of temperature and moisture are favorable. Infection may begin in the field through contact of the pods with the soil. Because of the slow development of Botrytis, the losses in transit are rarely ever large.

The study of the effect of temperature on infection and decay by *Botrytis cinerea* involved 3 experiments in which a snap bean was inoculated by placing a small bit of the fungus and agar in a slit made obliquely in the pod by means of a sterile knife. The inoculated pod was then placed at the center of a 4-qt. till basket of beans. In 2 of the experiments the quantity of beans at each temperature was limited to 1 basket and in the third to 2 and 3. The results given in table 16 are from the latter experiment. The variety Bountiful was used in each experiment.

Infection was obtained at a range of temperature from 0° to 35.5°. In 4 days some infection had taken place at 12°, 15.5°, 17.5°, 23°, 28°, and 35.5°. Judging by the number of infections at the various temperatures,

TABLE 16.—*Relation of temperature to infection of beans (Bountiful) by Botrytis cinerea*

Temperature	Relative humidity	Infection and decay after different periods of time in days									
		4[a]		6		8		11		15	
		Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected	Diameter of nest	Beans infected
°C.	*Per cent*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*	*Mm.*	*Number*
35.5	93	11	1.0								
28.0	95	9	0.5								
23.0	92	17	2.0								
17.5	89	21	3.5								
15.5	94	.....	2.5	38	10						
12.0	95	.....	1.0	20	3	45	10				
8.0	93	0	0.0	9	1	18	2	38	7		
8.0	98	0	0.0	9	3	.....	1[b]	30	2	45	10
2.0	96	0	0.0	0	0	0	0	.....	1	20	2
0.0	90	0	0.0	0	0	0	0	0	0	9	1

[a] The results on the fourth day or days are the averages of 2 baskets of beans at each temperature, those for the remaining period from 1 basket.

[b] The results at 6° were obtained from 3 baskets, after 6 days from 1, after 8 and 11 days from a second, and after 15 days from a third.

decay was mostly in the initial stages. In another experiment infection occurred at 22.5° in 2 days at at 6° and 8° in 6 days. The first infections observed at 2° occurred in 11 days and at 0° in 15 days. The maximum number of infections occurred at 17.5° in 4 days. In another experiment infection had occurred at 22.5° in 2 days, but none at the other temperatures. In the absence of more comprehensive results the optimum may be tentatively placed somewhere between 15.5° and 23°.

DETERIORATION OF SNAP BEANS NOT CAUSED BY DISEASE

A consideration of the conditions affecting development of transit diseases would be incomplete without some discussion of the effect of these conditions on the beans themselves.

Various signs of deterioration, such as bleaching, yellowing, rusting or bronzing, loss of turgor, and a consequent tendency to toughness, may develop during storage. These signs of deterioration lack sufficient definiteness to indicate their development very accurately. An effort, however, was made to characterize the loss of quality in the checks run parallel to the disease tests. (Tables 17 and 18).

In considering the results it should be kept in mind that the beans were from 4 days to possibly a week old when they were stored. It required from 2 to 4 days to transport the beans from Florida to Washington. The time between picking and loading undoubtedly varied, and the temperature and humidity in the railroad car during transit are unknown. It can be said, however, that they were sufficiently favorable to permit them to arrive in Washington in good condition.

The deterioration was rather rapid at the higher temperatures, taking place the most rapidly at 35° and 35.5°, the highest temperature employed, and decreasing in rate as the temperature was lowered. At 6° to 7° they retain their quality from 4 to 15 days. Below 6° the quality remained good from 6 to 15 days.

DISCUSSION AND CONCLUSIONS

In addition to the effects of temperature and humidity on infection and development of disease in beans *en route* to the market, important differences in diseases and in the pathogens that cause them must be taken into consideration in connection with any program dealing with the reduction of the losses in transit and marketing. Anthracnose and blight differ from the other diseases in being seed-borne, which involves the necessity of growing beans from seed free from these diseases. *Colletotrichum lindemuthianum, Pythium butleri, Rhizopus tritici, R. nigricans,* and *Botrytis cinerea,* reproduced by asexual spores can spread readily and rapidly. The asexual spores of the Rhizopus species and *B. cinerea* probably are spread more

TABLE 17.—*Deterioration of snap beans not caused by disease in various lots stored at different temperatures*

Experiment 1[a]				Experiment 2[b]				Experiment 3[c]			Experiment 4[d]			Experiment 5[e]				
Temperature	Relative humidity	Duration of storage	Color and condition	Temperature	Relative humidity	Duration of storage	Color and condition	Temperature	Relative humidity	Condition of beans after 3 days' storage	Temperature	Relative humidity	Condition of beans after 7 days' storage	Temperature	Relative humidity	Condition of beans after different periods of storage in days		
																4[f]	6	8
°C.	*Per cent*	*Days*		°C.	*Per cent*	*Days*		°C.	*Per cent*		°C.	*Per cent*		°C.	*Per cent*			
35.0	89	5	Yellow	35.5	93	4	Ripening and yellowing	33.6	91	Yellow to brown, very little snap								
33.0	84	5	"	31.0	95	4	"	29.0	96	Yellowing, very little snap	31.0	93	Yellow, maturing rapidly					
31.0	92	5	"	28.0	80	4	"	28.5	94	Slight yellowing, very little snap	29.0	97	Yellowish green to yellow	29.0	97	Yellowing	Yellowing	
28.0	83	5	"	24.5	94	4	"	25.0	93	Very slight yellowing very slight snap	25.0	94	Yellowish green	27.0	92	"	"	
23.0	88	5	Beginning to yellow	23.0	92	4	"	21.0	92	Color good as at start, snap slight	22.0	93	Pale green	25.0	95	"	"	
21.0	90	5	"	21.0	91	6	"	17.5	91	"	17.5	91	" "	22.0	93	"	"	
18.5	89	5	"	17.5	89	6	"	15.0	93	"	15.5	90	Pale yellow	17.5	87	"	"	
15.5	93	11	Considerable bleaching	15.5	94	6	"	12.5	94	"	12.5	93	" "	15.4	94	Normal	"	Yellowing

TABLE 17.—*Continued*

12.0	92	11	Slight bleaching	12.0	95	11	"	10.0	98	Color good, snap slight to fair	10.0	98	"	"	12.4	93	"	"	"
8.0	98	11	"	8.0	93	11	"	7.5	91	Color good, snap fair	7.0	91	Color and condition nearly as good as at start		10.0	98	"	Normal	"
6.0	98	11	Good condition	6.0	98	15	Good condition	2.0	97	"	2.0	97	"		7.0	93	"	"	"
2.0	98	11	"	2.0	97	15	"	1.3	95	Color good, snap excellent					6.0	95	"	"	Normal
				0.0	90	15	"												

[a] An unidentified variety shipped by express from Jensen, Florida, December 13, 1927. Arrived at Washington, D. C., Dec. 16, 1927. Stored December 17, 1927.

[b] Variety of beans used: Bountiful, from Ocala, Florida. Purchased on the Washington market, June 3, 1928. No other information regarding history of beans available. They were in good condition.

[c] Bountiful, shipped from Fort Lauderdale, Florida, January 23, 1930. Arrived at Washington, D. C., January 27 and stored January 28, 1930. Good condition.

[d] Bountiful. Shipped from Indiantown, Florida, February 22, 1930. Arrived at Washington, D. C., February 25 and stored February 26, 1930. Condition good.

[e] Bountiful. Shipped from Delray Beach, Florida, May 10, 1930. Arrived at Washington, D. C., and stored May 13, 1930.

[f] This lot of beans was also inspected after 2 days of storage, when, except for slight flabbiness at the higher temperatures, it was in good condition.

TABLE 18.—*Physiological deterioration of snap beans as related to temperature and time*[a]

Temperature	Relative humidity	Condition of beans after different periods of time in days							
		1	2	3	4	6	7	8	11
° *C.*	*Per cent*								
35.0	92	Marked yellowing	Marked yellowing	Marked rusting	Light green to rusty	Yellow and rusty	Yellow to rusty		
31.0	92	Considerable discoloration	Considerable discoloration	"	"	Light green to rusty	"		
28.5	95	"	"	Considerable discoloration	Light green	Considerable rusting	"		
25.0	91	Very slight discoloration	"	"	" "	Light green to rusty	Light yellow to rusty		
19.5	94	No discoloration	Slight discoloration	Slight discoloration, ripening	" "	Light yellow	Light yellow		
17.5	91	"	Very slight discoloration	Very slight discoloration	" "	Considerable bleaching	" "		
15.0	94	"	"	"	Slight bleaching	"	Light green to yellow		
12.0	93	"	No discoloration	Very slight bleaching	Very slight bleaching	"	"		
10.0	99	"	"	"	"	Slight bleaching	"		
7.0	93	"	"	No discoloration	No discoloration	Very slight bleaching	Very slight bleaching	Slight bleaching	Slight bleaching and rusty
2.0	97	"	"	"	"	No discoloration	"	"	"

[a] Two varieties of beans were used in these experiments. Bountiful, shipped from Fort Lauderdale, Florida, February 23, 1931, arrived at Washington February 25, 1931, and Giant Stringless Green Pod, shipped from Canal Point, Florida, February 24, 1931. Both varieties were stored at the various temperatures February 27, 1931.

readily than those of *C. lindemuthianum* and *P. butleri*. *Sclerotinia sclerotiorum*, *Sclerotium rolfsii*, and *Rhizoctonia solani* do not produce viable asexual spores at all, and thus are more limited in their capacity to spread. The vegetative nature of *Bacterium phaseoli* is such as to permit rapid spread. *S. sclerotiorum*, *R. tritici*, *R. nigricans*, and *P. butleri* reproduce sexually, and, in case of the last 3, oospores are produced that serve as resting bodies to bridge the fungi over unfavorable conditions. *S. sclerotiorum*, *S. rolfsii*, *R. solani*, and *B. cinerea* form sclerotial bodies that aid in perpetuating their existence from season to season. The sclerotia of all 4 organisms germinate directly into a vegetative condition. The sclerotia of *S. sclerotiorum* may germinate directly or produce apothecia whose ascospores facilitate the spread of the pathogen. In addition to being carried over from season to season in the seed, the blight pathogen is thought to be able to live over winter on bean refuse, especially in the warmer climates. The other pathogens are all able to live over in the soil. (*C. lindemuthianum* is probably short-lived in the soil.) *S. sclerotiorum*, *P. butleri*, and *S. rolfsii* are primarily soil organisms, and most of the primary infections probably take place by the pods coming in contact with the soil. Beans may become infected by *B. cinerea* through their contact with the soil, but infection usually takes place in transit as a result of the pods becoming contaminated with spores.

The host range of anthracnose and blight, as reported, is limited to the common bean and a few closely related species and genera. None of the susceptible cultivated crops are likely to be grown in rotation with beans. *Sclerotium rolfsii* attacks a variety of cultivated crops (beet, cabbage, corn, eggplant, potato, pepper, radish, sweetpotato, tomato, etc.) and is widely distributed throughout the South. *Sclerotinia sclerotiorum*, widely distributed in the United States, attacks a number of vegetable crops (cabbage, carrot, celery, cauliflower, eggplant, lettuce, etc.), some of which are often grown in rotation. *Rhizoctonia solani* attacks potatoes, turnips, beets, alfalfa, lettuce, cabbage, etc., and is widely distributed. *Pythium butleri* affects such crops as cucurbits, eggplant, and tomato. *Rhizopus tritici*, *R. nigricans*, and *Botrytis cinerea* are more or less ubiquitous and omnivorous and will attack beans in transit whenever conditions are favorable. These pathogens are less dependent on field infection and contamination than the others.

It has been assumed throughout these experiments that a high humidity in the bean container is favorable to infection and decay by the various pathogens. A relative humidity of 90 per cent and above has been provided in nearly all cases. Because of the high water content of beans, a high humidity is probably nearly always present in the shipping container. In some instances, especially when the beans are packed wet and when the

fluctuation of temperature is such as to bring about condensation, free water will be found on them in the container.

Wounding is not essential to infection of pods detached from the vines unless it be in the case of blight.

Major emphasis should be directed towards the field control of anthracnose, bacterial blight, watery soft rot, southern blight, soil rot, and cottony leak. Effective control measures are available for the first 2 diseases. All diseased beans should be sorted out and the sound beans packed when dry and kept as cool as possible. If such beans are shipped at temperatures below 12°, very little, if any, decay by Rhizopus soft rot and cottony leak would take place. There would be little, if any, loss from anthracnose at temperatures of 10° and below in less than 10 or 11 days, and none at all at temperatures below 7°. The lowest temperature at which infection by *Sclerotium rolfsii* has been obtained is 8° in 11 days. Infection by *Rhizoctonia solani* took place in 5 days at 10.5°, in 16 days at 8° and 5.5°, and in 20 days at 0.9° and 2°.

The most difficult diseases to limit and control during transit are gray-mold rot and watery soft rot. Fortunately, because of its slow rate of development, gray-mold rot is not the source of heavy losses. If beans are contaminated with *Sclerotinia sclerotiorum*, the best that can be done is to retard its development by shipping them at temperatures (0° to 5°) as low

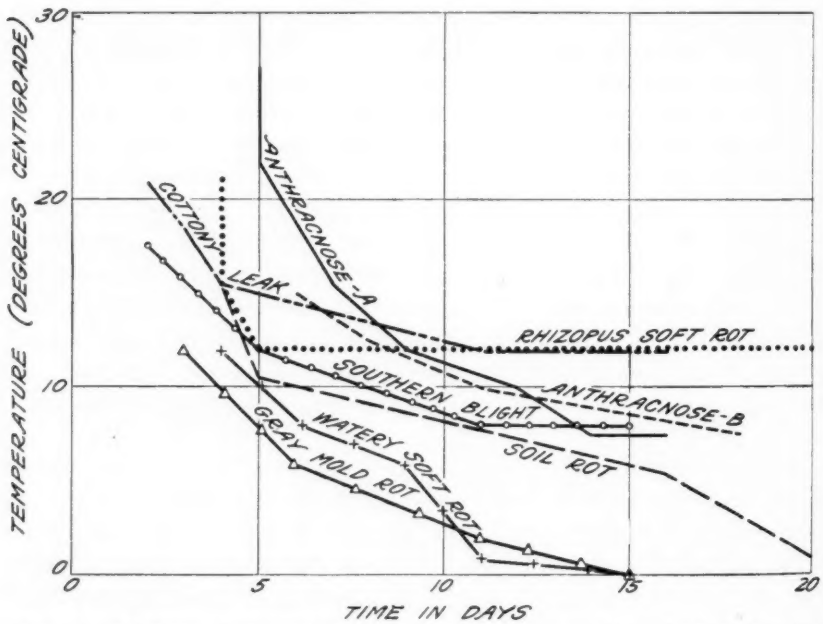

FIG. 4. Minimum time required at certain temperatures either for infection to occur or incipient infection of various diseases to become visible on beans.

as possible without incurring the danger of freezing. One bean had become infected with *S. sclerotiorum* from one point of inoculation in 4 days at 12°, 5 pods in 6 days at 8°, and infection had just started in 11 days at 2°, and in 15 days at 0°. Figure 4 illustrates the minimum time required for infection to occur at a number of temperatures in the case of all diseases except blight. In plotting these curves only cases of initial infection were used, and the graphs are based on all the results of all the experiments.

Taking into consideration the effects of the different temperatures on the quality of the beans as well as on the development of the diseases, the lower the temperature (above the freezing point of beans) employed during shipment the better the results will be.

SUMMARY

Colletotrichum lindemuthianum will infect not only wounded but also wound-free beans when the pods are sprayed with a water suspension of spores. Infection took place in beans inoculated by means of a needle prick at temperatures from 8° to 33° and in beans inoculated by spraying with a spore suspension at temperatures from 7° to 27°. Lesions have developed in visibly healthy pods sorted out from stock showing the presence of anthracnose, when stored at temperatures from 7.5° to 29°. The optimum temperature for infection ranges from 22° to 25°, the highest percentage usually occurring at 25°. The shortest time in which infection has been observed to occur at temperatures of 22°, 25°, and 27° was 5 days; at 15.5° and 17.5°, 7 days; at 12°, 9 days; at 10°, 12 days; and at 7°, 14 days. *C. lindemuthianum* spores obtained from living bean pods seem to be more virulent than those obtained from artificial cultures.

All attempts to obtain infection by spraying snap beans with a water suspension of *Bacterium phaseoli* were unsuccessful. Infections through needle pricks were obtained at temperatures from 2° to 31°. Development of blight lesions at temperatures from 1.2° to 35.0° were observed in beans free from visible signs of infection, which were sorted out from lots showing the presence of blight. The development was very slight at 35° and rather slow at temperatures of 7° and below. There was little difference in the amount of blight that developed at 10° to 24.5°.

Infection of snap beans by *Sclerotinia sclerotiorum* was obtained at temperatures from 0° to 28°. The time required for infection to occur at temperatures from 15.5° to 27.5° was not accurately determined. Five to 15 beans had been infected from one point of inoculation in 4 days at 3 temperatures. The earliest infection at 12° was observed in 4 days, at 8° in 6 days, at 5.5° to 6° in 9 days, at 1.8° to 2° in 11 days, and at 0° in 15 days. The optimum temperature for infection is approximately 19° to 24°.

The range of temperatures at which infection of snap beans has been obtained by *Pythium butleri* extends from 12° to 35.6°. Infection at 12° and 15.5° was limited to inoculated beans after 16 days' storage. The optimum temperature for infection is about 31°.

Rhizoctonia solani infected snap beans at 0.9° to 35.5°. Twenty days were required for infection to start in the inoculated beans at 0.9° and 2°, and 16 days at 5.5° and 8°. The optimum temperature for infection extends from 24° to 32°.

Infection by *Sclerotium rolfsii* was obtained on snap beans at temperatures from 8° to 35.6°, the latter being near the maximum. Infection at 8° and 12° was limited to the inoculated pod after 11 days of storage. No infection occurred at temperatures below 8°.

Infection by *Rhizopus tritici* and *R. nigricans* is not dependent on artificial inoculation and will occur to some extent in snap beans exposed to temperatures above 30°. Infection has been observed in both inoculated and noninoculated beans at temperature extending from 12° to 35.5°. Infection below 25° is usually very slight. No infection has ever been obtained at temperature below 12°.

Botrytis cinerea has infected snap beans at temperature from 0° to 35.5°. Infection occurred at 12°, 15.5°, 17.5°, 23°, 28°, and 35.5° in 4 days. Judging by the amount present at the various temperatures, infection was still in the initial stages. Infection took place at 6° and 8° in 6 days, at 2° in 11 days, and at 0° in 15 days.

Deterioration of snap beans, aside from that produced by microorganisms, is very rapid at temperatures above 20°, especially at 35.5°, and decreases with the lowering of the temperature.

In snap beans shipped from Florida to Washington, D. C., the quality remained good for 4 to 15 days at temperatures from 6° to 7° and for 6 to 15 days at temperatures below 6°.

Taking into consideration both the quality of snap beans and the diseases likely to occur in transit, shipment at temperatures below 10° is desirable and the lower the temperature (above the freezing point of beans) the better.

BUREAU OF PLANT INDUSTRY,
WASHINGTON, D. C.

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SOME VIRUSES AFFECTING SPINACH, AND CERTAIN ASPECTS OF INSECT TRANSMISSION[1]

ISMÉ A. HOGGAN

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The mosaic disease of spinach (*Spinacia oleracea* L.) known as "spinach-blight" has occupied a unique position among plant virus diseases for some time, as a result of the reported transmission of the causal agent of the disease from infective parent aphids to their progeny through several generations (13). This constitutes the only evidence yet recorded that tends to indicate a hereditary association of a plant virus with the transmitting insect. Since other investigations have revealed no further evidence of a similar nature, there appears to be some question how far plant viruses may be regarded as "inheritable" by offspring of infective insect vectors. The subject is of sufficient general interest and importance in its bearing on various problems of plant-virus transmission to merit further attention, and any information tending to clarify the present situation should be of some value.

In the course of recent investigations, it has been shown (7) that the cucumber-mosaic virus is transmissible to spinach, producing symptoms on this host that closely resemble those of spinach-blight, according to the description of the latter disease by McClintock and Smith (13). On the basis of this similarity in symptoms, as well as in aphid vectors, the suggestion was made that the two diseases might be identical, and that, if this were so, further light might be thrown on the insect relationships of the spinach-blight virus. It was, therefore, decided to undertake a more critical investigation of cucumber mosaic on spinach and of its possible connection with spinach-blight, in the hope of contributing further information on the "inheritability" of this disease by the aphid vectors. Unfortunately, however, attempts to secure material of spinach-blight from the district where it was originally described have met with little success, so that it has been necessary to resort to more indirect methods in comparing the two diseases.

Since Böning (2) has recently concluded that a mosaic of spinach occurring in Germany, which he appears to regard as identical with the American spinach-blight, is probably due to the virus of beet mosaic, the present investigation has been extended to include a comparative study of this virus also in relation to spinach. Some additional work has been carried out with a third virus transmissible to this host plant, namely, that of

[1] Cooperative investigations of the Wisconsin Agricultural Experiment Station and the Division of Tobacco and Plant Nutrition, Bureau of Plant Industry, U. S. Department of Agriculture.

tobacco ring-spot (5, pp. 16–17). These 3 viruses have been studied primarily from the standpoint of a possible connection with spinach-blight, though certain other considerations have arisen during the investigation that have widened the scope of the work. These include problems of comparative means of transmission, of the development of local as contrasted with systemic infection, and of certain relationships between virus and aphid vector.

PREVIOUS INVESTIGATIONS

That spinach-blight, as it occurred in Virginia, was an infectious disease of the mosaic type was conclusively demonstrated by McClintock and Smith (13) in 1918. These investigators showed the disease to be readily transmissible from plant to plant by needle inoculation with diseased plant extract, as well as by several species of aphids, of which *Myzus persicae* and *Macrosiphum solanifolii* appeared to be the most important. They divided the course of the disease into 8 arbitrary stages, the characteristic symptoms of which may be summarized as follows: 1. A very slight yellowing of the younger leaves of infected plants and occasionally of one or more older leaves. 2. More pronounced yellowing of the younger leaves. 3. Malformation of the younger leaves, which become much wrinkled, narrowed, and mottled; yellowing of the older leaves and some stunting of the plant. 4. Distinct stunting of the plant, with cessation of growth; yellowing of the entire foliage; mottling of the older leaves and extreme malformation of the younger ones, which are very finely savoyed and feathery in appearance. 5. Browning and death of parts of the older leaves, usually progressing from the outer tips inwards. 6. Total disintegration of the older leaves and some browning of the central ones. 7. Browning of the younger leaves. 8. Death of the plant, which has become reduced to a very small central whorl of leaves.

The same authors presented evidence believed to indicate a direct transmission of the virus from one generation of infective aphids to another. This evidence was based in large part upon the rearing of colonies from infective parent aphids on certain host plants other than spinach, such as lettuce, pepper, eggplant, and potato, and the transfer of individuals from succeeding generations produced on these hosts back to healthy spinach. In nearly all cases, one or more of the spinach plants so treated became diseased, while needle inoculations from the foliage of the previous host of the aphids gave no infection. Similarly, transfers to healthy spinach of aphids naturally present in the field on such plants as celery, sweet potato, and wild mustard resulted in some infection with spinach-blight. In another experiment, young of virus-bearing aphids, *Macrosiphum solanifolii*, were transferred at birth to 25 healthy spinach plants, one of which became

diseased. Since the aphids causing these infections had at no time come in contact with diseased spinach, these results were interpreted as indicating a "hereditary" association of the virus with the insects.

The failure to secure infection of spinach by artificial inoculation from the leaves of the lettuce and other plants on which the infective aphid colonies were reared led the authors to conclude that it was improbable that such plants served as alternate hosts of the virus. No further conclusions were reached, however, as to the host range of the disease.

Later, L. B. Smith (18) succeeded in developing a variety of spinach that, although not strictly immune from the disease, showed a high degree of resistance to it in the field. This variety was named "Virginia Savoy."

A mosaic of spinach prevalent in Germany has been described in recent years by various writers under the name "*Mosaikkrankheit*" or "*Gelbfleckigkeit*" (3, 2, 21, 22). The characteristic symptoms of this disease are a yellowing and necrosis of the foliage, accompanied by severe stunting or death of the plant. According to Volk (21), plants infected when young rapidly become yellow all over and die, while older plants show first a curling and yellowing of the younger leaves, the discoloration then spreading over the foliage and the plant becoming much stunted. Later, the outer leaves turn brown and die, leaving a few dwarfed yellow-green leaves in the center. Eventually, the whole plant succumbs.

The opinion appears to be general among the German investigators that this disease is identical with the American spinach-blight. Thus, Schaffnit (3, p. 173), referring to the investigations of McClintock and Smith, states: "*. . . die von den Autoren* [McClintock and Smith] *beschriebene Krankheit zweifellos mit der bei uns vorkommenden identisch ist.*" Volk (21, p. 1) and Wichmann (22, p. 160) later make similar statements. This conclusion appears to be based upon a general agreement in symptoms between the 2 diseases and in transmissibility by aphids and by artificial inoculation. According to Böning (2, p. 492), however, the leaf symptoms at first consist of a number of small, usually round, yellow spots about the size of a pin's head ("*so erscheint die Spreite wie übersät mit vielen, meist runden Fleckchen von etwa Stecknadelkopfgrösse*"), and this symptom is clearly illustrated in figure 3 of his paper. This point will be referred to again later; for the present it should be noted that McClintock and Smith do not mention any discrete spotting as an early symptom of spinach-blight. Böning adds that the spots are sometimes so numerous as to coalesce, forming larger yellow areas. Of the leaf malformations described by McClintock and Smith he observed a puckering of the leaf surface and a narrowing of the lamina, particularly in the female plants in the flowering stage.

The German disease was shown to be transmissible to healthy spinach plants by aphids (*Aphis fabae* Scop. and *Macrosiphum* sp.), and less read-

ily by artificial inoculation (2). Böning was also able to transmit this mosaic to beet, and beet mosaic to spinach, by means of aphids, the symptoms developing on the respective hosts being the same for either disease. He, therefore, concluded that the two diseases were probably identical. In this connection the reader is reminded that a mosaic of both garden and sugar beet has been reported at different times by various investigators in Europe and in America (14, 4, 12, 19, 16, 1, 11).

MATERIALS AND METHODS

Upon first suspecting a relationship between cucumber mosaic and spinach-blight, attempts were made to secure material of the latter disease from Virginia and from other sections of the United States for purposes of comparison with cucumber mosaic. In spite of repeated efforts, however, no such material was obtained until the investigation was nearly completed. It, therefore, became necessary to establish certain arbitrary criteria by which the degree of resemblance of each virus under consideration to that of spinach-blight might be evaluated. The salient characteristics of the spinach-blight virus, according to the descriptions of McClintock and Smith (13), appeared to be the following: (1) Ready transmission from plant to plant both by aphids (particularly *Myzus persicae* and *Macrosiphum solanifolii*) and by artificial inoculation (needle-prick method) and (2) a characteristic sequence of symptoms on affected plants. This symptom sequence has been summarized earlier. The investigation has, therefore, consisted mainly in transmission and symptomatology studies of the respective viruses on spinach. A third criterion was based upon the resistance of Virginia Savoy spinach to spinach-blight, this variety being tested for indications of resistance to the different viruses under consideration.

Sugar-beet plants affected with mosaic were kindly furnished by P. N. Annand, Twin Falls, Idaho, and by C. W. Bennett, Riverside, California. This disease appeared to be identical with the sugar-beet mosaic described by Robbins (16). The cucumber-mosaic virus studied (*cucumber virus 1* (10)) was the same as that employed in previous transmission studies (6, 7, 8), having been obtained originally from a mosaic cucumber plant; and the tobacco ring-spot virus was derived from material received from S. A. Wingard, Virginia Agricultural Experiment Station, Blacksburg, Virginia. Seed of Virginia Savoy spinach was obtained through the courtesy of F. P. McWhorter, Virginia Truck Experiment Station, Norfolk, Virginia.

The investigation was carried out entirely in the greenhouse. Two methods of virus transmission were employed: (1) Aphid transmission by means of *Myzus persicae* and *Macrosiphum solanifolii* and (2) artificial inoculation of plant extract. The methods involved in the colonizing and handling of the aphids were similar to those employed in previous trans-

mission studies and have been described in detail elsewhere (6, 8). Virus-free colonies of *Myzus persicae* were maintained on cabbage and those of *Macrosiphum solanifolii* on potato. In aphid-transmission trials, it was customary to transfer to the control plants a number of nonviruliferous aphids direct from the stock colonies or from other healthy plants.

Two methods of artificial inoculation were employed. In the "cotton-needle" method, a small quantity of absorbent cotton was wrapped around the tip of an ordinary dissecting needle, and this was dipped into the infectious plant extract and used to scratch and prick the leaf lamina and midrib. The second method was essentially the rubbing method of Holmes (9), the leaf surface being wiped with a piece of cheesecloth moistened with virus extract. Samuel (17) has recently pointed out that this is a very effective means of securing infection with several viruses. With smooth-leaved plants, such as sugar beet and spinach, however, a heavy rubbing that bruised without actually tearing the leaf, amounting almost to a "leaf mutilation" inoculation, proved more effective than a light wiping and was practiced throughout for smooth-leaved plants. For convenience, both variations of this method will be referred to as the "rubbing" method, following Holmes's terminology. Plants serving as controls in artificial inoculation series usually received no treatment.

The following host plants were used during the investigation: Tobacco, *Nicotiana tabacum* L., Havana Seed type; spinach, varieties Bloomsdale and Virginia Savoy; sugar beet, *Beta vulgaris* L., unknown variety; and cucumber, *Cucumis sativus* L., variety Select White Spine. Vigorous seedlings were used in all trials. The spinach and sugar beet were grown in a cool greenhouse (65–70° F.), the tobacco and cucumber in a warmer house (80–85° F.). Most of the work involving spinach was conducted in the early spring and fall months, when light and temperature conditions favored good growth. This plant made very poor growth during the winter and tended to rapid flower-stalk formation during the longer and warmer days of late spring and summer.

Each transmission trial was repeated several times, a total of 20 plants usually being tested for each type of virus transfer. It was believed that the results obtained in this way, from several repetitions of each experiment, were more significant than when fewer trials were made, each involving larger numbers of plants.

EXPERIMENTAL RESULTS

The suggestion advanced by Böning (2) of a probable relationship between spinach mosaic and beet mosaic immediately raised the question of the specificity of the virus or viruses responsible for the latter disease. So far as could be judged from the description of symptoms of both beet mo-

saic and sugar-beet mosaic published by different European and American investigators (14, 16, 1, 11), it appeared likely that the same virus was involved in most, if not all, cases. Almost no information was available, however, on the host range of the disease or on any other diagnostic characters of the virus beyond the fact that it was transmitted by aphids (16, 1). Since cucumber mosaic was found to be transmissible to sugar beet, it seemed conceivable that the cucumber-mosaic virus might be identical with that causing sugar-beet mosaic. While this subsequently proved not to be the case, a comparison of the two viruses on sugar beet appeared to be a necessary preliminary to the main investigation.

Comparison of Viruses on Sugar Beet and Tobacco. The cucumber-mosaic virus was found to be readily transmissible from tobacco to sugar

TABLE 1.—*Transmission of the cucumber mosaic virus to sugar beet and spinach*[a]

Mosaic host (source of virus)	Host	Mode of transmission							
		Myzus persicae		*Macrosiphum solanifolii*		Needle inoculation		Rubbing inoculation	
		Infested	Control	Infested	Control	Inoculated	Control	Inoculated	Control
Tobacco	Sugar beet	30/30	30/0	20/20	20/0	20/0	20/0	20/0(19)	20/0
	Spinach var. Bloomsdale	30/28	30/0	20/20	20/0	20/19	20/0	20/20	20/0
	Spinach var. Virginia Savoy	40/17	40/0	40/19	40/0	20/0	20/0	20/0	20/0
Sugar beet	Tobacco	20/19	20/0	20/17	20/0	20/9	20/0	20/20	20/0
Spinach var. Bloomsdale	Spinach var. Bloomsdale	20/20	20/0	25/25	25/0	30/28	30/0	20/20	20/0
	Tobacco	20/20	20/0	20/17	20/0	20/9	20/0	20/20	20/0
Spinach var. Virginia Savoy	Spinach var. Virginia Savoy	20/15	20/0	20/10	20/0	20/0	20/0	20/0	20/0
	Tobacco	20/14	20/0	20/8	20/0	20/7	20/0	20/20	20/0

[a] "Infested" denotes plants to which aphids were transferred from a mosaic host; "inoculated" denotes plants artificially inoculated with virus extract; and "control" denotes plants serving as controls to those either infested or inoculated. In all fractions, the numerator represents the total number of plants infested with aphids, artificially inoculated or treated as controls as the case may be; the denominator, the number of plants becoming infected. Figures in parentheses denote plants developing local infection only.

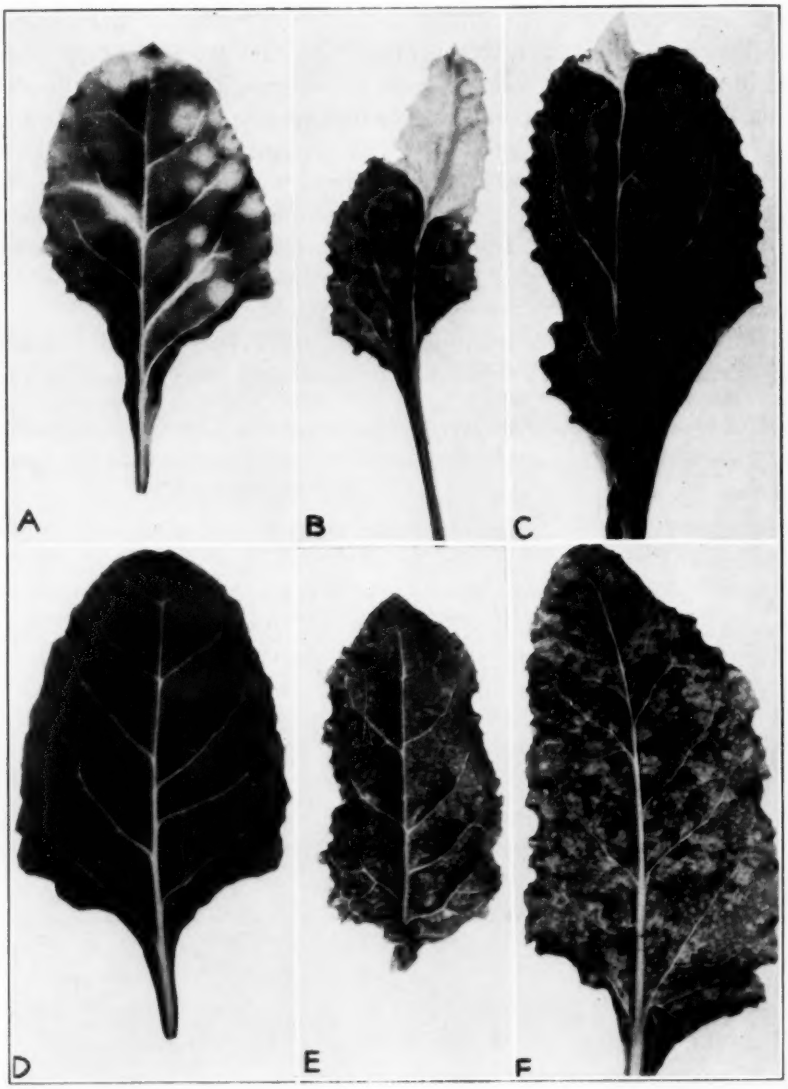

FIG. 1. Leaf symptoms produced by viruses on sugar beet. A. Local lesions of cucumber mosaic transmitted by *Myzus persicae*. B and C. Cucumber mosaic. Chlorosis due to systemic infection. D. Leaf of normal plant. E. Sugar-beet mosaic, early symptoms of systemic infection. F. Sugar-beet mosaic, later symptoms.

beet, and *vice versa*, by means of the aphids *Myzus persicae* and *Macrosiphum solanifolii* (Table 1), species that have been shown elsewhere to be efficient vectors of this virus to other hosts (6, 8). When, following the usual procedure in aphid-transmission trials, viruliferous aphids were al-

lowed to feed freely on the foliage of young sugar-beet plants systemic infection of the plants was readily obtained. The first sign of infection, however, was the appearance of conspicuous, chlorotic spots on the infested leaves, 5 or more days after the transfer of viruliferous aphids to the plant (Fig. 1, A). These spots were evidently of the nature of "local lesions" and appeared to develop at points of introduction of the virus into the tissues by the aphids. They were never observed on leaves on which the aphids were known not to have fed nor on leaves on which only nonviruliferous aphids had fed. When first observed, the lesions were pale yellow green, usually circular, though sometimes irregular in form, 2 mm. or more in diameter, and generally with a very diffuse margin. They grew rapidly larger, becoming brighter yellow and frequently coalescing to form irregular, rather indefinite, chlorotic areas. Sometimes a minute necrotic dot was visible at the center of the spot.

Symptoms of systemic infection became apparent one or more days after the appearance of the local lesions. These consisted essentially in a chlorosis of the whole or part of the newly developing leaves, successive leaves generally showing progressively smaller chlorotic areas, situated at the apical end of the leaf (Fig. 1, B, C). Chlorosis of this type is characteristic also of the earlier stages of infection of tobacco with the same virus. Occasionally, dark green, savoyed areas were present on such leaves. Leaves developing later showed a coarse mottling of relatively large light and dark areas, accompanied by considerable stunting of the plant. Plants thus infected continued to put forth mottled leaves throughout the season's growth. The virus was readily recovered from such plants onto tobacco both by means of aphids and by the rubbing method of artificial inoculation, and less readily by needle inoculation of extracted juice (Table 1).

Systemic infection of sugar beet with the cucumber-mosaic virus was not secured, however, by either method of artificial inoculation (Table 1). Local symptoms, more or less similar to the local lesions resulting from infection by aphids, were usually observed on leaves that had been inoculated by the rubbing method,[2] and the virus was later recovered from these leaves by means of inoculation of extracted juice to tobacco. Since the cucumber-mosaic virus is known to lose its infectivity within a few days outside the living host, it is evident from these results that the virus had been able to establish itself in the inoculated leaves. No virus was recovered at any time, however, from *uninoculated* leaves of the same plants, even after 3 or more weeks had elapsed; in other words, there was no systemic infection of plants following artificial inoculation.

[2] Samuel (17, p. 502) has also reported local infection of sugar beet with the cucumber-mosaic virus, following a similar method of inoculation.

A similar condition obtained when viruliferous aphids, instead of being allowed to wander freely over the plant, were confined to a single leaf within a glass tube closed at both ends. Local lesions, resembling those previously described, developed under these conditions on the infested leaf, enlarging and coalescing until the whole leaf became yellow and eventually died. The virus was readily recovered from such leaves by the rubbing method of inoculation. However, no systemic infection of the plants followed, all uninfested leaves remaining normal in appearance and free from virus, as tested by subsequent inoculations to tobacco.

Explanation of this peculiar situation is at best only tentative. For some reason, under the conditions of the experiment, the cucumber-mosaic virus appears unable to pass from a single infected leaf of this host to other parts of the plant. The systemic infection obtained when the aphids were not restricted to any particular organ may possibly have resulted from a direct introduction of virus at or near the growing point of the shoot, the virus perhaps multiplying at this point and forming a source of infection for all subsequent growth. In this connection it was observed that the bud was a favorite feeding point of the aphids.

Comparison of the symptoms of cucumber mosaic on sugar beet with those of sugar-beet mosaic soon showed that the two diseases were not the same, although both proved transmissible by the same species of aphids. Sugar-beet mosaic has been reported by Robbins (16) to be transmissible by *Myzus persicae*, and in the present investigation ready transmission of the virus was obtained on sugar beet by means of this aphid, and also with *Macrosiphum solanifolii*. With either species, 100 per cent infection was secured in all trials, all control plants remained healthy (Table 2). The virus was found transmissible also by artificial inoculation, particularly when a severe rubbing method was used. Thus, of 20 plants tested by either method, 18 were infected by rubbing and 10 infected by needle inoculation, systemic infection developing in all cases, while all control plants remained healthy (Table 2). In this respect the virus differed from that of cucumber mosaic, with which systemic infection of sugar beet was not secured through artificial inoculation.

It may be noted that several previous investigators (16, 1, 11) have reported failure to obtain transmission of beet mosaic or of sugar-beet mosaic by methods of artificial inoculation. Assuming the same virus to be involved in all cases, this apparent inconsistency in results may no doubt be ascribed to differences in the type of technique employed. Verplancke (20), on the other hand, claims to have transmitted beet mosaic by rubbing the leaves of healthy plants with mosaic-plant extract.

Systemic infection with sugar-beet mosaic resulted also when viruliferous aphids were confined to single leaves of healthy sugar-beet plants. This

TABLE 2.—*Transmission of the sugar-beet mosaic virus to sugar beet and spinach*[a]

Mosaic host (source of virus)	Host	Mode of transmission							
		Myzus persicae		*Macrosiphum solanifolii*		Needle inoculation		Rubbing inoculation	
		Infested	Control	Infested	Control	Inoculated	Control	Inoculated	Control
Sugar beet	Sugar beet	20/20	20/0	20/20	20/0	20/10	20/0	20/18	20/0
	Spinach var. Bloomsdale	20/20	20/0	20/20	20/0	20/15	20/0	20/20	20/0
	Spinach var. Virginia Savoy	20/20	20/0	20/19	20/0	20/13	20/0	20/18	20/0
	Tobacco	20/0(20)	20/0	10/0(10)	10/0	20/0	20/0	20/0	20/0
Spinach var. Bloomsdale	Spinach var. Bloomsdale	20/20	20/0	20/17	20/0	20/12	20/0	20/16	20/0
	Sugar beet	20/20	20/0	20/20	20/0	20/7	20/0	20/17	20/0
Spinach var. Virginia Savoy	Spinach var. Virginia Savoy	20/20	20/0	20/19	20/0	20/12	20/0	20/17	20/0
	Sugar beet	20/20	20/0	20/19	20/0	20/9	20/0	20/20	20/0

[a] See footnote to table 1.

contrasts again with the behavior of the cucumber-mosaic virus, which caused only local infection under similar conditions, and would seem to indicate some distinct difference in the host-tissue relationships of the respective viruses.

Since the symptoms of sugar-beet mosaic have been described in detail by Robbins (16), only the more important distinguishing features will be considered at the present time. On young sugar-beet plants, the first symptoms observed were usually those of systemic infection; though under certain conditions, in aphid transmission trials, these were preceded by the appearance of faint chlorotic spots on the infested leaves, presumably arising at points where the aphids had fed. These lesions were about 1 mm. in diameter and remained much smaller and less conspicuous than those associated with local infection of the same host with cucumber mosaic.

Systemic infection became apparent in the form of minute yellow or light green flecks on the young leaves 5 or more days after infection (Fig. 1, E). These flecks were less than 1 mm. in diameter and were situated for

the most part over the veins. Sometimes they were so numerous as to give temporarily the effect of a marked clearing of the veins. They increased somewhat in size, the mosaic pattern soon becoming resolved into a sharply defined, fine, speckly mottling, quite different from the chlorosis or coarse, indefinite mottling characteristic of cucumber mosaic on this host. Leaves of later development usually showed a similar type of mottling (Fig. 1, F), though sometimes larger light and dark areas were formed. These differences in the symptoms produced by the 2 viruses should be clear from a comparison of the illustrations to which reference has been made.

A further comparison of the 2 viruses was made on tobacco, a host already known to be susceptible to cucumber mosaic. No infection of tobacco with the sugar-beet-mosaic virus was obtained, however, by either method of artificial inoculation. On the other hand, when attempts were made to transfer this virus by means of aphids, certain symptoms developed on the infested leaves of the tobacco plants, apparently at points where the aphids had fed, although systemic infection did not follow. These local symptoms consisted of small, round, light brownish yellow spots on the leaf lamina, 1 to 2 mm. in diameter, with very diffuse margins and often with a minute necrotic point in the center (Fig. 2, A). That these represented centers of multiplication of the sugar-beet-mosaic virus within the leaf was indicated by the fact that the virus could readily be recovered from such leaves by rubbing inoculation of extracted juice to healthy sugar beet. Moreover, tobacco plants infested with nonviruliferous aphids from healthy sugar beet showed no spotting. The lesions were first visible 3 or more days after infestation of the plants with infective aphids and enlarged quite rapidly to about 1 cm. in diameter, later becoming gradually less distinct. Sometimes they coalesced to form a diffuse yellowish pattern on the leaf. The spots showed a marked tendency to necrotic ring formation, often developing 2 or more fine, concentric rings (Fig. 3). Similar, though less numerous, lesions were produced on tobacco by viruliferous individuals of *Macrosiphum solanifolii*. On the other hand, all uninfested leaves of the same tobacco plants remained normal in appearance and free from all symptoms of disease, nor could any virus be recovered from them, even several weeks after the appearance of the lesions on infested leaves. It was, therefore, concluded that systemic infection had not taken place. The symptoms produced by this virus on tobacco are entirely distinct from those of cucumber mosaic, with which systemic infection is readily secured both by aphids and by artificial inoculation, resulting in a very different type of chlorosis and mottling of the leaves.

From this preliminary comparison of cucumber mosaic and sugar-beet mosaic it is concluded that the 2 diseases differ considerably from one an-

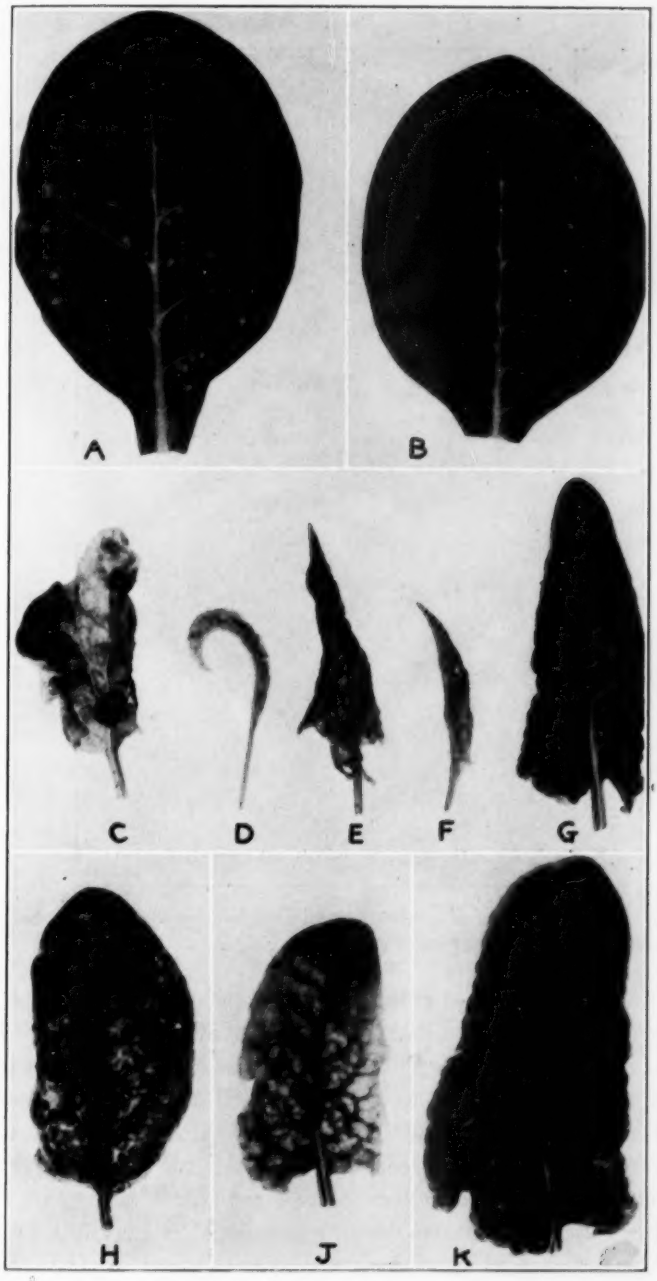

FIG. 2. Leaf symptoms produced by viruses on tobacco and spinach (variety Bloomsdale). A. Local lesions of sugar-beet mosaic on tobacco, following transmission by *Myzus persicae*. B. Leaf of normal tobacco plant. C–F. Cucumber mosaic on spinach, showing mottling and malformation of young leaves. G. Corresponding leaf of normal spinach plant. H. Early symptoms of sugar-beet mosaic on spinach. J. Tobacco ring-spot on spinach. K. Corresponding leaf of normal spinach plant.

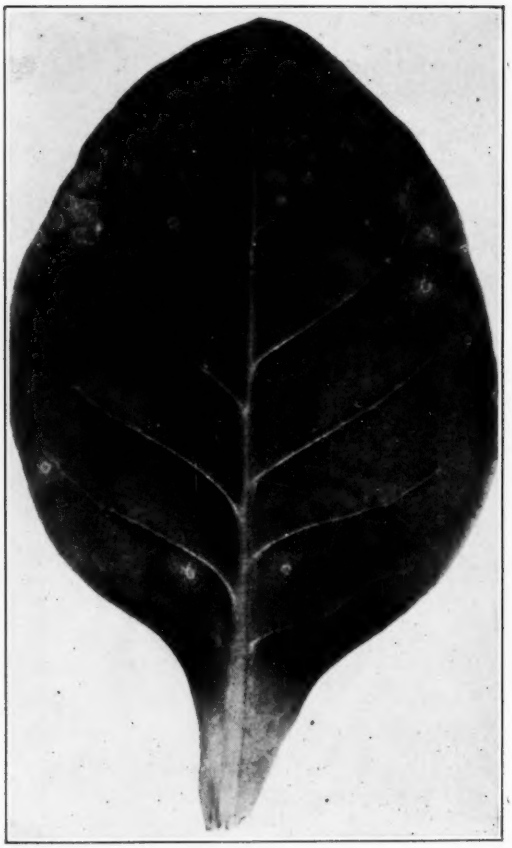

FIG. 3. Local lesions of sugar-beet mosaic on tobacco resulting from infection by *Myzus persicae*, showing development of necrotic rings.

other in their behavior and symptoms, both on sugar beet and on tobacco, and it seems clear that 2 quite distinct viruses are concerned.

In this connection it may be well to summarize briefly the characteristic symptoms on these 2 host plants of the third virus under consideration, namely, that of tobacco ring-spot. On tobacco, the ring-spot virus typically produces an intricate necrotic ring and line pattern, consisting of concentric rings and parallel zigzag lines, sometimes accompanied by mottling of the leaf and associated with systemic infection of the plant (23). On sugar beet, Wingard (23) reports the development of faint zigzag lines on the leaves, of lighter color than the normal tissue, but not necrotic. Similar symptoms, representing systemic infection, have been observed on this host by the writer, though in some cases the lines became necrotic.

Comparison of Viruses on Spinach. Following the preliminary comparison of viruses on sugar beet and tobacco, it was now possible to proceed to a study of their symptoms and behavior on spinach, with the object of comparing each virus with that causing spinach-blight. As previously indicated, the chief known characteristics of the spinach-blight virus appeared to be its ready transmission both by aphids and by artificial inoculation, its symptom sequence on spinach, and the resistance to infection manifested by the Virginia Savoy variety.

The results of transmission trials with the cucumber-mosaic virus on spinach are summarized in table 1. Throughout this investigation the variety Bloomsdale has been used as representing a variety of spinach susceptible to spinach-blight. When this variety was tested, the cucumber-mosaic virus was found to be very readily transmissible from tobacco to spinach and from spinach to spinach by means of either *Myzus persicae* or *Macrosiphum solanifolii* and also by both needle and rubbing methods of inoculation. With each method of transmission, 100 per cent infection was almost always obtained. The virus was also readily recovered from Bloomsdale spinach onto tobacco by means of aphids and inoculation by rubbing and less readily by needle inoculation. In all trials, the control plants remained free from infection.

The sequence of symptoms of cucumber mosaic on Bloomsdale spinach, occurring on young plants under the greenhouse conditions prevalent during the investigation, was found to correspond remarkably closely to that described for spinach-blight by McClintock and Smith (13). The symptoms developed gradually, the incubation period varying greatly according to the time of year and the prevailing environmental conditions. The minimum time observed between inoculation and the first appearance of recognizable symptoms was 7 days for infection by aphids and 12 days for artificial inoculation. This corresponds with the minimum period of 7 days mentioned by McClintock and Smith for spinach-blight. As with their disease, however, incubation periods of 2 weeks or more were quite common under certain conditions of light and temperature.

The first recognizable symptom produced by the cucumber-mosaic virus was a very faint, general chlorosis, sometimes involving the entire foliage, sometimes, especially with older plants, apparent only on the younger leaves. As growth continued, this chlorosis became more intense and spread to the outer leaves also, if these were not originally affected, until at a later stage of the disease the entire plant became bright yellow. At the same time the newly developing leaves began to show evidence of malformation in varying degrees. In milder cases, this malformation consisted in a reduction in width of the lamina, frequently accompanied by considerable wrinkling and an inward rolling of the leaf margin (Fig. 2, E, F).

In more severe cases, some leaves became reduced to a fine ribbon (Fig. 2, D); others were much puckered, twisted, or distorted; while some developed a mottling with marked savoying of the darker areas (Fig. 2, C). Similar types of malformation have been described by McClintock and Smith (13) in association with spinach-blight. As the yellowing and malformation progressed, the plant became much stunted and growth was increasingly retarded. A plant at this stage, showing discoloration of the entire foliage and slight malformation of the inner leaves, with marked inward rolling of the margins, is illustrated in figure 4, A. Later, a progres-

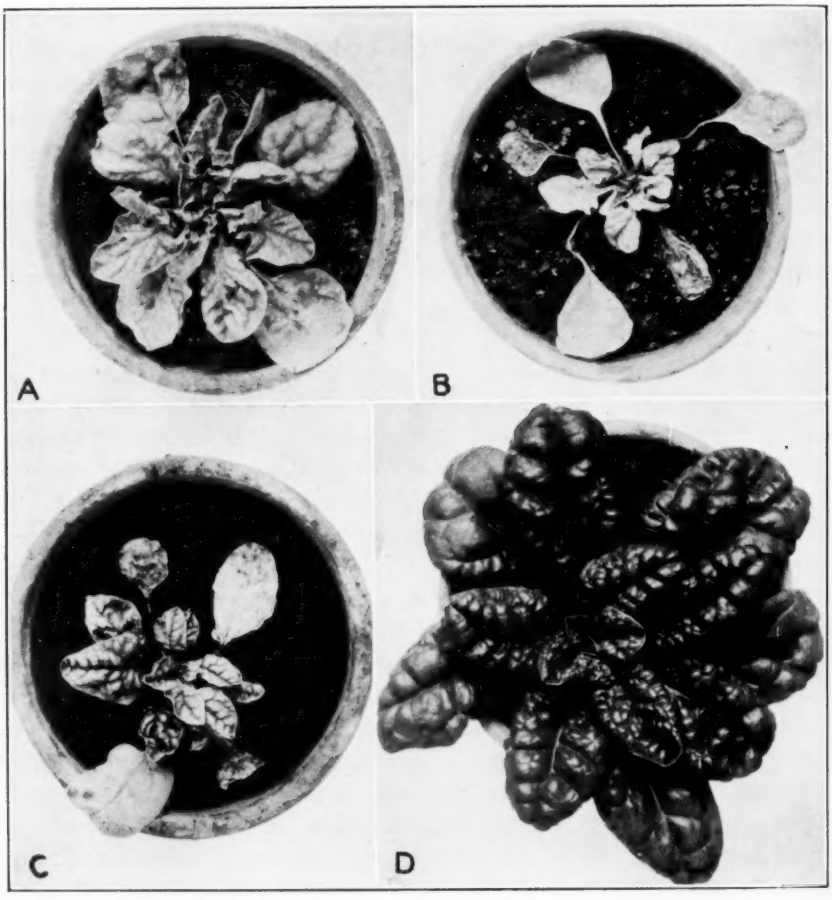

FIG. 4. Symptoms produced by viruses on spinach (variety Bloomsdale). A. Cucumber mosaic, moderately advanced stage of disease. B. Cucumber mosaic, later stage, showing necrosis of outer leaves. C. Tobacco ring-spot, severe infection. D. Normal plant of approximately the same age as plants A and C.

sive necrosis of the leaves developed, involving first the outermost whorl and slowly working inwards. As the affected leaves turned brown and shriveled and growth declined, the plant became gradually reduced in size (Fig. 4, B), until only a small central cluster of yellow, malformed leaves remained. Finally the whole plant died. These symptoms, both in character and sequence, are essentially the same as those described by McClintock and Smith for spinach-blight (13).

In tests of the susceptibility of the Virginia Savoy variety to cucumber mosaic, variable results were obtained, depending apparently upon the season and the resulting environmental conditions. At times it was found possible to secure a high percentage of infection of this variety by means of aphids, while at other times, following an identical procedure, no infection of the plants took place. Thus, of a total of 80 Virginia Savoy plants tested at various times with aphids from cucumber-mosaic tobacco, 17 out of 40 became infected by means of *Myzus persicae* and 19 out of 40 by *Macrosiphum solanifolii* (Table 1). On the other hand, parallel trials performed simultaneously with Bloomsdale spinach invariably yielded extremely high percentages of infection with aphids under all conditions. Similarly, transmission of the virus by aphids could be secured at times from Virginia Savoy to Virginia Savoy, though less consistently than with Bloomsdale. Moreover, in all earlier trials, artificial inoculation of Virginia Savoy, by either needle or rubbing method, failed to yield infection of any plants, whether diseased tobacco or Virginia Savoy was used as the source of inoculum; although in parallel trials, conducted at the same time, Bloomsdale was readily infected by these means. Thus, of a total of 40 Virginia Savoy plants tested for either method of artificial inoculation all remained healthy (Table 1). At the close of the investigation, however, in a repetition of these trials, some infection of very young Virginia Savoy plants was obtained through the rubbing method of inoculation, tobacco being used as the source of inoculum. These results are not included in table 1. In further trials the virus was readily recovered from diseased Virginia Savoy by means of aphids and rubbing inoculation and less readily by the needle method, all control plants remaining free from infection throughout. From these results it is, therefore, concluded that, although Virginia Savoy may be susceptible to cucumber mosaic under certain greenhouse conditions, this variety shows much greater resistance to the disease than does the variety Bloomsdale.

The onset of symptoms of cucumber mosaic was much the same on Virginia Savoy as on Bloomsdale, though in the former variety the yellowing and necrosis usually progressed much more quickly, necrosis of the bud soon setting in, resulting in rapid death of the plant (Fig. 5, C). Consequently, as a rule, very little growth was made after the first appearance

of symptoms, and the stage of malformation of newly developing leaves did not always occur. Older plants, however, which usually died less rapidly, developed very conspicuous malformation of the young leaves, these being reduced to small, yellow, almost ribbon-like structures.

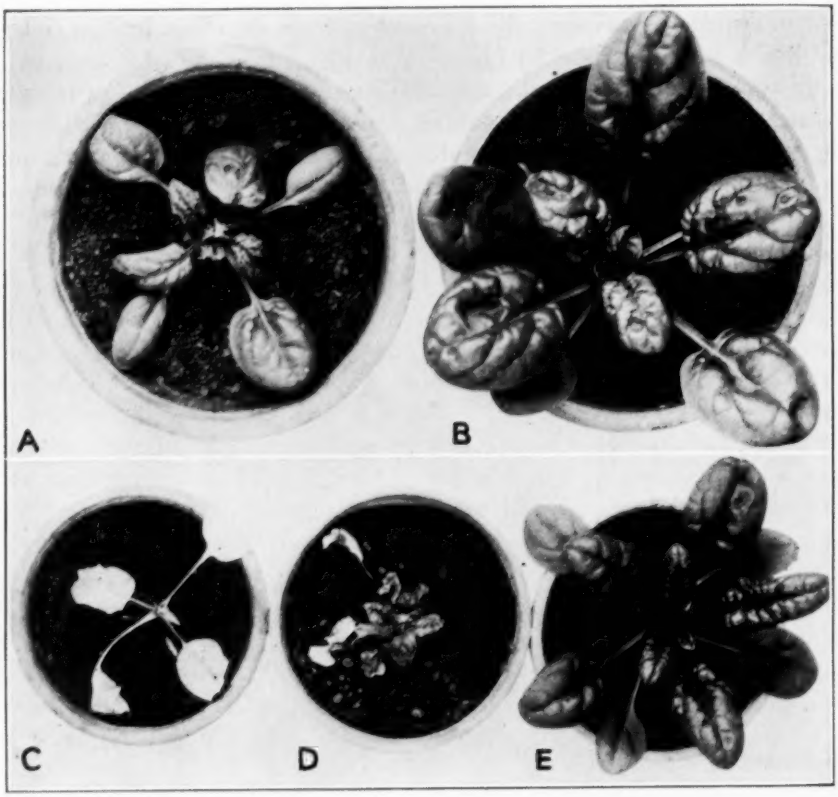

FIG. 5. Symptoms produced by viruses on spinach (variety Virginia Savoy). A. Sugar-beet mosaic, early stage of disease. B. Normal plant, control to A. C. Cucumber mosaic transmitted by *Myzus persicae*, showing rapid death of plant. D. Sugar-beet mosaic, late stage of disease. E. Normal plant, control to C and D.

The actual factors governing resistance or susceptibility of Virginia Savoy to infection with cucumber mosaic have not been determined, although there was some indication that both air temperature and light intensity might influence the amount of infection secured and possibly also the size of the plants at the time of inoculation. It should be pointed out that Virginia Savoy is not reported as immune from spinach-blight, though it exhibits a relatively high degree of resistance to the disease in the field as compared with other commercial varieties (18). In this respect, there-

fore, there appears to be considerable resemblance between the virus causing spinach-blight and that of cucumber mosaic. It has already been shown that the 2 diseases are strikingly similar in symptom sequence and also in ease of transmission on suspectible varieties, both by artificial inoculation and by aphids. While similarity in symptoms alone is not to be regarded as conclusive evidence of the identity of 2 viruses, this factor, when coupled with close agreement in other respects as well, should afford a more reliable indication of relationship. On the basis of these considerations, therefore, there appears to be very strong evidence in favor of the conclusion that the 2 diseases are very closely related, if not identical. On the other hand, sugar-beet mosaic on spinach, while resembling spinach-blight in some respects, shows also some distinct differences, both in its symptom complex and in the lack of any indications of resistance to the disease in the Virginia Savoy variety.

The results of transmission trials with sugar-beet mosaic on spinach are included in table 2. As with cucumber mosaic, the virus was found to be readily transmissible to Bloomsdale spinach and from spinach to spinach by means of both species of aphids and inoculation by rubbing. It appeared, however, to be somewhat less readily transmissible from plant to plant by needle inoculation than was the cucumber-mosaic virus, 60 to 75 per cent infection with sugar-beet mosaic being obtained by this method of inoculation as compared with 94 to 95 per cent infection with cucumber mosaic. Moreover, the sugar-beet-mosaic virus proved to be as readily transmissible to Virginia Savoy, from both infected sugar beet and Virginia Savoy, as it was to Bloomsdale, by means of aphids and by artificial inoculation, although in parallel trials with the cucumber-mosaic virus little or no infection of Virginia Savoy might be secured. Thus, there was no indication of any resistance in Virginia Savoy to sugar-beet mosaic, a point that suggests a distinct difference between this disease and spinach-blight. In further trials, the virus was readily recovered onto sugar beet from both varieties of spinach by means of aphids and by rubbing inoculation and less readily by needle inoculation. In all trials with this virus, all control plants remained free from infection.

The symptoms of sugar-beet mosaic on spinach appeared to be identical on both Bloomsdale and Virginia Savoy, and the following description of the disease is applicable to either variety. As with cucumber mosaic, the incubation period varied greatly according to the time of year and the prevailing environmental conditions. The minimum period observed was 6 days for infection by aphids and 7 days for artificial inoculation, although periods of 2 to 3 weeks or more were quite common under less favorable conditions.

The onset of symptoms of sugar-beet mosaic on spinach was quite sudden. The first sign of infection was usually a marked downward arching

of the young leaves, accompanied or soon followed by the appearance of numerous bright golden yellow flecks on the young leaves (Figs. 2, H, and 5, A). These flecks were round or irregular in form, less than 1 mm. in diameter, often with a minute darker point in the center. Frequently they became so numerous as to coalesce, forming large yellow areas. There was no faint uniform chlorosis of the foliage at this stage, as there was with cucumber mosaic, but always a distinct spotting, of a type not mentioned by McClintock and Smith as an early symptom of spinach-blight, although very similar to the mottling described and illustrated by Böning (2) as an early symptom of the spinach mosaic occurring in Germany. Indeed, the symptoms illustrated in figure 3 of Böning's paper (2) appear almost identical with those of sugar-beet mosaic on spinach, as shown in figure 2, H, of the present paper. Leaves of later development showed similar symptoms and were considerably dwarfed and puckered, although undergoing no marked malformation such as has been described for spinach-blight (13). In this connection it may be noted that Böning (2), while discussing the symptoms of the German spinach mosaic, describes, as types of malformation observed, only a puckering of the leaf and a narrowing of the lamina, particularly in older plants at the flowering stage.

As the disease progressed, the yellow coloration gradually spread over the foliage, the plant became much stunted, and growth soon ceased. Necrosis of the outer leaves then developed, as with cucumber mosaic, the leaves usually dying back from the tip. This necrosis spread slowly inward until the plant became reduced to a central whorl of small, mottled, and puckered leaves (Fig. 5, D) and finally died. Plants of the Bloomsdale variety, especially when very young, usually succumbed more rapidly to sugar-beet mosaic than to cucumber mosaic, except at higher temperatures, when the symptoms of sugar-beet mosaic were less severe and the plants tended to survive. At certain stages, however, there might be a decided resemblance between the 2 diseases, especially on older plants, and, if the malformation associated with cucumber mosaic was not marked, it sometimes required considerable experience to distinguish between the 2 on the basis of symptoms alone.

From the foregoing description it will be seen that sugar-beet mosaic on spinach, while closely resembling spinach-blight in certain respects, differs from it in the occurrence of a fine, discrete spotting as an early symptom of the disease and in the absence of any marked malformation of the leaves of seedling plants such as is associated with "spinach-blight. When one considers also the less ready transmissibility of the virus on spinach by needle inoculation and the fact that Virginia Savoy shows no evidence of resistance to sugar-beet mosaic, it becomes apparent that this disease bears less resemblance to the originally described spinach-blight than does

cucumber mosaic. On the other hand, sugar-beet mosaic on spinach resembles so closely the mosaic of spinach described by various German authorities (3, 2, 21, 22) as to suggest a probable identity of these 2 diseases, thus supporting the contention of Böning (2) that the spinach mosaic prevalent in Germany is identical with beet mosaic.

When the tobacco ring-spot virus was found to be transmissible to spinach, it was thought that some comparative tests of the symptoms and behavior of this virus also on spinach might be of interest, since the disease is known to be prevalent on other hosts in certain districts. Only a few trials were conducted, however, as it was soon shown to differ in several respects from spinach-blight.

Tobacco ring-spot is known to be a disease of wide host range, readily transmissible by certain methods of artificial inoculation (23). Up to the present, however, no insect vectors of the virus have been determined. In the family Chenopodiaceae, garden and sugar beet are known to be susceptible to the disease (15, 23), though, so far as the writer is aware, spinach, *Spinacia oleracea* L., has not previously been recorded as a host of the virus.

The tobacco ring-spot virus proved very readily transmissible from tobacco to spinach and from spinach to spinach (var. Bloomsdale) by both rubbing and needle methods of artificial inoculation (Table 3). In limited tests, Virginia Savoy appeared to be equally susceptible to infection by these means. The virus was also readily recovered from Bloomsdale spinach to tobacco through inoculation by rubbing, although infection was

TABLE 3.—*Transmission of the tobacco ring-spot virus to spinach*[a]

Mosaic host (source of virus)	Host	Mode of transmission							
		Myzus persicae		*Macrosiphum solanifolii*		Needle inoculation		Rubbing inoculation	
		Infested	Control	Infested	Control	Inoculated	Control	Inoculated	Control
Tobacco	Spinach var. Bloomsdale					15/15	15/0	15/15	15/0
	Spinach var. Virginia Savoy					10/10	10/0	10/10	10/0
Spinach var. Bloomsdale	Spinach var. Bloomsdale	20/0	20/0	20/0	20/0	10/10	10/0	10/10	10/0
	Tobacco	10/0	10/0	10/0	10/0	10/0	10/0	10/10	10/0

[a] See footnote to table 1.

not obtained in this transfer by means of needle inoculation. No infection resulted in any trials following attempts to transmit the virus by aphids (*Myzus persicae* and *Macrosiphum solanifolii*); in this respect the virus differed markedly from that of spinach-blight.

The minimum incubation period observed on spinach was 9 days. The first symptoms were those of systemic infection, large chlorotic areas appearing on the young leaves, sometimes becoming bright yellow (Fig. 2, J). These areas showed a marked tendency to necrosis, the entire affected region usually drying out very rapidly. No malformation of the leaves was observed. The plants showed a tendency to recover, especially in warm weather, subsequently developing leaves often being almost free from symptoms. Death of the plant did not occur under the conditions prevailing during the trials, although occasionally the entire foliage became yellow and the plant much stunted (Fig. 4, C). This disease, therefore, did not appear to bear a very close resemblance to spinach-blight. It is significant, however, that all 3 viruses studied in the present connection characteristically produce conspicuous yellowing and necrosis of the leaves of this host and that 2 at least may cause death of the plant.

Isolation of Cucumber Mosaic from Naturally Infected Spinach. Toward the close of the investigation here reported, a number of spinach plants of a Savoy type, said to be naturally infected with spinach-blight, were kindly sent by E. E. Clayton from the Long Island Vegetable Research Farm of the New York Agricultural Experiment Station, Riverhead, New York. Ten of these plants were selected at random and attempts were made to isolate from them any virus or viruses responsible for the diseased condition. This was accomplished by means of a transfer of aphids from each plant to healthy Bloomsdale spinach and, later, as soon as symptoms of disease appeared, from these spinach plants to tobacco and to sugar beet. Artificial inoculations were made also from the original spinach plants to tobacco and sugar beet. By these means a virus was recovered from all 10 plants whose symptoms on the various hosts closely resembled those of cucumber mosaic. A direct comparison was, therefore, made of the symptoms produced by this virus on tobacco and on cucumber with those of known cucumber-mosaic virus (*cucumber virus 1* (10)) by means of parallel inoculations to young cucumber and tobacco plants. On each host, the symptoms produced by either virus were almost identical. A comparison was made also of the thermal death point and longevity *in vitro* of either virus in tobacco-plant extract. In these determinations the thermal death point for each virus was found to lie between 65 and 70° C. and the longevity *in vitro* between 1 and 2 days. On the basis of these results, the virus isolated from the spinach plants was, therefore, concluded to be *cucumber virus 1*. No sugar-beet-mosaic virus was recovered from any of

the plants, as was indicated by the absence of any symptoms of this virus on any of the hosts inoculated; nor was the presence of any other virus detected. Unfortunately, no further material of spinach-blight has been secured at any time, so that too much significance should not be attached to these results. They show, however, that cucumber mosaic may affect spinach in the field, producing symptoms characteristic of spinach-blight, and thus support the conclusion that true spinach-blight and cucumber mosaic are both due to the same virus.

"Inheritability" of the Cucumber-mosaic Virus by Progeny of Infective Aphids. Having concluded that the original spinach-blight is probably identical with cucumber mosaic on spinach, it seemed desirable to make some test of the capacity of the aphid vectors of cucumber mosaic for direct transmission of the virus from one generation of insects to the next in some such manner as had been done for spinach-blight.

McClintock and Smith (13) performed 2 types of experiment in this connection. In the first type, colonies of *Myzus persicae* and *Macrosiphum solanifolii*, originating from viruliferous stock, were bred through several generations on hosts supposedly not susceptible to spinach-blight and were tested at intervals for infectivity by transfers to healthy spinach. In the second type, young of infective *M. solanifolii* were transferred at birth to healthy spinach before they had time to feed upon the mosaic host.

Since the writer has not been able to discover any host plant suitable for colonization of either species of aphid that is not susceptible to infection with cucumber mosaic, either locally or systemically, it was decided to repeat the second type of experiment, using *Macrosiphum solanifolii* as the test species. The following procedure was adopted. Colonies of the aphid were bred for several generations exclusively on cucumber-mosaic spinach of the variety Bloomsdale before being tested in the experiment. All plants used as mosaic hosts showed characteristic symptoms of the disease in moderately advanced stages of infection. Young of adult aphids born and reared on such plants were then removed at birth by means of a fine camel's-hair brush and placed on healthy spinach plants in insect-proof cages. While McClintock and Smith transferred on an average 2 aphids to each plant, it seemed preferable in this test to use a larger number per plant, since previous experience with the transmission of the same virus by the peach aphid had indicated that infection was not regularly obtained with such small numbers of insects.[3] Ten nymphs were, therefore, transferred to each of 15 plants, so that in all 150 young were tested for infectivity. The aphids were closely observed after transference in order to ascertain whether they were in any way injured by handling, any individuals showing

[3] Hoggan, Ismé A. Some factors involved in aphid transmission of the cucumber-mosaic virus on tobacco. (In manuscript.)

any signs of injury being promptly replaced. The aphids were allowed to remain on the plants for a week or more, during which time they made apparently normal development. They were then destroyed by fumigation and the plants observed for signs of infection. As a test of the infectivity of the parent aphids, 10 adults were transferred to each of 15 plants and treated in a similar manner. Fifteen untreated spinach plants served as a control. All plants were observed over a period of 5 weeks before final records were made. Of the 15 plants to which the young nymphs had been transferred, all remained perfectly healthy in appearance throughout the experiment, while of the 15 plants infested with adult nymphs, 8 developed typical symptoms of the disease in less than 3 weeks. Although this does not represent a particularly high percentage of infection, it is approximately what would be expected on the basis of previous results obtained on the transmission of the cucumber-mosaic virus by varying numbers of peach aphid.[4] All control plants remained healthy throughout. There was, therefore, no evidence in this experiment of any direct transmission of the virus from infective parent aphids to their progeny.

The test was not repeated with *Myzus persicae* since a similar experiment, shortly to be reported elsewhere,[5] has been carried out with this species, using cucumber-mosaic tobacco as host. It may be stated, however, that here again there was no indication of any inheritance of the virus by progeny of infective parents.

Properties of the Sugar-beet-Mosaic Virus. Since an effective method of artificial transmission of sugar-beet mosaic was available in a modification of Holmes's rubbing method of inoculation (9), it was thought that a determination of some of the properties of the virus in mosaic-sugar-beet extract might be of value as an aid to later identification. The following properties were studied: longevity *in vitro,* tolerance to dilution, and thermal death point.

All virus extracts were prepared by macerating the leaves of mosaic sugar-beet plants in a meat grinder and straining the juice through cheesecloth. Extractions were made soon after characteristic symptoms of the disease had developed on the plants. Inoculations were made to young sugar-beet plants by rubbing 3 leaves of each plant with a piece of cheesecloth dipped in virus extract until the leaves were considerably bruised, usually the 3 lowest leaves exclusive of the cotyledons being selected. The hands were disinfected between each set of inoculations by washing thoroughly with soap and water. Inoculations from untreated extract, which served as a control, were always made last. Four separate determinations were made for each property, 5 plants in each test being inoculated with each sample of extract.

[4] See footnote [3].

[5] See footnote [3].

In the tests of longevity *in vitro,* 2 cc. of virus extract was placed in each of several test-tubes and stored in the dark at an air temperature of about 70° F. The contents of each tube were aged for a different period of time and separate inoculations made from each. For determinations of the thermal death point, 2 cc. of freshly extracted juice was heated for 10 minutes in a thin-walled test-tube in a water-bath maintained at the desired temperature, then cooled rapidly in cold running water. Dilutions were made by adding 5 cc. of freshly extracted juice to 45 cc. of distilled water, then transferring 5 cc. of the mixture to 45 cc. of distilled water, and so on. In this way dilutions of 1 in 10, 1 in 100, etc., were obtained.

The results of trials of longevity *in vitro* are given in table 4. While

TABLE 4.—*Longevity in vitro of the sugar-beet-mosaic virus*

Age of extract in hours	Number of plants infected of 5 inoculated				Total number of infections (20 plants)
	Trial 1	Trial 2	Trial 3	Trial 4	
0 (fresh extract)	5	5	5	5	20
3	5	3	4	3	15
6	4	2	4	2	12
9	3	3	4	2	12
12	1	3	4	1	9
18	1	2	3	1	7
24	0	1	1	0	2
48	0	0	0	0	0
Uninoculated controls	0	0	0	0	0

some variation occurred between individual trials, it will be seen that the percentage of infection gradually decreased with aging, until after 24 hours the extracts yielded only 2 infections on a total of 20 plants. Extract aged for 48 hours gave no infection in any trial. According to these determinations, therefore, the longevity of this virus *in vitro,* at a temperature of about 70° F., should be placed at 24–48 hours.

The tests of tolerance to dilution (Table 5) yielded consistent results. In these tests there was a marked drop in the percentage of infection secured at a dilution of 1 in 100, and extracts diluted to 1 in 1,000 gave only 2 infections on a total of 20 plants. At higher dilutions, no infection was obtained. The tolerance to dilution was, therefore, concluded to be 1 in 1,000.

TABLE 5.—*Tolerance to dilution of the sugar-beet-mosaic virus*

Dilution of extract	Number of plants infected of 5 inoculated				Total number of infections (20 plants)
	Trial 1	Trial 2	Trial 3	Trial 4	
None	5	5	5	5	20
1 in 10	4	3	4	4	15
1 in 100	1	1	1	1	4
1 in 1,000	0	1	1	0	2
1 in 10,000	0	0	0	0	0
Uninoculated controls	0	0	0	0	0

A preliminary test of the thermal death point of the virus indicated that this probably lay somewhere between 50 and 60° C. for a 10-minute exposure. Tests were, therefore, run at 50, 55, and 60° C., and a separate extraction of juice was made for each exposure in order to avoid any effect of aging, since the virus had already been shown to lose some of its infectivity within a few hours *in vitro* (Table 4). As a control, unheated extract was allowed to stand for 10 minutes at room temperature before inoculation, and this proved to be highly infectious in all trials.

The results of 4 separate experiments are presented in table 6. In each test extract heated at 50 and 55° C. yielded some infection, while extract

TABLE 6.—*Thermal death point of the sugar-beet-mosaic virus (10-minute exposure)*

Temperature in °C.	Number of plants infected of 5 inoculated				Total number of infections (20 plants)
	Trial 1	Trial 2	Trial 3	Trial 4	
50	5	2	3	2	12
55	1	1	2	2	6
60	0	0	0	0	0
Unheated extract	5	4	5	5	19
Uninoculated controls	0	0	0	0	0

heated at 60° C. gave none. The thermal death point of the virus was, therefore, concluded to be between 55 and 60° C.

DISCUSSION

Evidence has been presented above pointing to the conclusion that the disease originally described as "spinach-blight" is identical with cucumber mosaic on spinach. The wide range of hosts of the cucumber-mosaic virus and the cultivation of several of these in some of the spinach-growing districts of the United States, together with the ready transmission of the virus by various species of aphids, would no doubt be sufficient to account for the epidemics of the disease that occurred in certain regions before the introduction of resistant varieties of spinach. It is unfortunate, however, that direct comparisons have not been possible with spinach-blight from districts where this disease is, or has been, most prevalent. Final test of relationship should rest upon a relatively simple matter of inoculation from affected plants to one or more characteristic hosts of the cucumber-mosaic virus; though admittedly there might still be insufficient foundation for the assumption that a disease now recognized as spinach-blight was actually identical with the trouble with which McClintock and Smith dealt 15 years ago. The demonstration that spinach is susceptible to several distinct viruses having certain characteristics in common suggests the possibility that more than one virus disease of this host may have been included at one time or another under the name spinach-blight, although it appears most likely that the disease originally described by McClintock and Smith (13) was strictly due to a single virus. L. K. Jones (11) reports the occurrence of beet mosaic on spinach in Washington, and it is possible that the crop may be subject to different virus diseases in different parts of the country. Moreover, if the conclusions reached in the present paper are correct, the spinach mosaic described in Germany would appear to be due to the same virus as sugar-beet mosaic, as originally suggested by Böning (2), but not to be identical with the spinach-blight of McClintock and Smith. Such considerations emphasize again the need of a critical analysis of the specific virus or viruses concerned in any particular disease, and the value of determinations of differential characters other than symptoms as additional means of diagnosis.

If one accepts the conclusion that the original spinach-blight and cucumber mosaic on spinach are identical, another interpretation might be applied to certain results obtained by McClintock and Smith, believed to indicate a direct transmission of the virus from one generation of aphids to another. Cucumber mosaic is now known to have a wide host range and to affect such plants as potato, eggplant, and pepper, hosts used by these investigators for the breeding of successive generations of aphids from infective parents. Certain other hosts from which the aphids were tested, namely, lettuce, celery, and mustard, have also been found by the writer to be susceptible

to this disease. Although McClintock and Smith obtained no infection of spinach by needle inoculation from several of the hosts upon which the aphids had bred, in the light of present knowledge it seems possible that such plants may actually have served as sources of virus for successive generations of aphids. Furthermore, no evidence has been obtained in connection with the cucumber-mosaic virus that the young of infective parent aphids are themselves infective before they have fed on a mosaic host. In view of the great fundamental significance attaching to any fully authenticated case of direct inheritance of a plant virus by progeny of an infective insect vector, it is, therefore, felt that particular caution should be exercised in accepting such an interpretation of results in the absence of further confirmation.

Other points of interest that have arisen during the investigation may barely be touched upon at the present time. The curious behavior of the cucumber-mosaic virus on sugar beet resulting in systemic or only localized infection according to the mode of introduction of the virus into the plant suggests specific relationships with the various tissues of the host about which much remains to be learned. The differential behavior of the cucumber- and sugar-beet-mosaic viruses with respect to the development of local or systemic infection both on sugar beet and on tobacco might be utilized as a means of separation of either virus from a combination of the two. Attention is directed also to the fact that, while previous studies on "local lesions" have dealt mainly with those induced by artificial inoculation, the present investigation affords examples of similar lesions resulting from infection by aphids and representing feeding points of viruliferous insects.

SUMMARY

Spinach, *Spinacia oleracea*, is shown to be susceptible to at least 3 different virus diseases, all of which characteristically cause conspicuous yellowing and necrosis of the foliage, while 2 of them frequently result in death of the entire plant. These diseases are: cucumber mosaic, sugar-beet mosaic, and tobacco ring-spot. That each of these diseases is due to a distinct, specific virus is concluded from a comparison of their symptoms and behavior on certain common hosts.

Cucumber mosaic is shown to be transmissible to sugar beet, systemic or localized infection resulting according to the mode of transmission employed. The development of local lesions as a result of infection by aphids is described in the case of cucumber mosaic on sugar and of sugar-beet mosaic on tobacco.

Cucumber mosaic is very readily transmissible to spinach (var. Bloomsdale) by artificial inoculation and by the aphids *Myzus persicae* and *Macrosiphum solanifolii*. The symptom sequence on this host closely resembles

that of spinach-blight as originally described by McClintock and Smith. Virginia Savoy spinach, known to be resistant to spinach-blight, shows definite indications of resistance to cucumber mosaic under greenhouse conditions.

Spinach plants obtained from Long Island, said to be naturally infected with spinach-blight, were shown to be infected with the virus of cucumber mosaic (*cucumber virus 1*).

Sugar-beet mosaic is readily transmissible to spinach by means of aphids and by artificial inoculation, the Virginia Savoy variety showing no indications of resistance to the disease under greenhouse conditions. The symptoms on spinach differ in several respects from those described for spinach-blight, although closely resembling those of a mosaic of spinach present in Germany.

Tobacco ring-spot is readily transmissible to either variety of spinach by artificial inoculation, though the resulting symptoms bear no very close resemblance to those of spinach-blight. No transmission of the virus was obtained by means of aphids.

Determination of certain properties of the sugar-beet-mosaic virus, made as an aid to later identification, indicates that the longevity *in vitro* is between 24 and 48 hours at a temperature of about 70° F., the tolerance to dilution 1 in 1,000, and the thermal death point for a 10-minute exposure between 55 and 60° C.

On the basis of the extreme similarity in symptom sequence between cucumber mosaic on spinach and the spinach-blight of McClintock and Smith, together with their ready transmission by artificial inoculation and by aphids, and the resistance to either disease manifested by the Virginia Savoy variety, it is suggested that the 2 diseases are identical. On the other hand, a mosaic of spinach described in Germany and considered by some to be identical with the American spinach-blight is believed to be due to the virus of sugar-beet mosaic.

No indication was obtained of any direct transmission of the cucumber-mosaic virus from infective parent aphids, *Macrosiphum solanifolii*, to their progeny. The evidence previously presented as indicating an inheritance of the spinach-blight virus by successive generations of aphids is discussed in the light of the possible relationship between this disease and cucumber mosaic, and reasons are advanced for not accepting this evidence as conclusive in the absence of further confirmation.

UNIVERSITY OF WISCONSIN,
MADISON, WISCONSIN.

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RECORDS OF CITRUS SCAB MAINLY FROM HERBARIUM SPECIMENS OF THE GENUS CITRUS IN ENGLAND AND THE UNITED STATES

ANNA E. JENKINS AND H. S. FAWCETT[1]

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INTRODUCTION

For the purposes of obtaining more information on the occurrence and distribution of the disease known as citrus scab, material of the genus Citrus (mostly cultivated) was examined in 1930 at the herbaria of the Royal Botanic Gardens, Kew, and the Department of Botany, British Museum, South Kensington, London, England, and in 1931, at the Gray Herbarium, and Arnold Arboretum, Harvard University, Cambridge, Massachusetts; New York Botanical Garden, New York, N. Y.; the Philadelphia Academy of Natural Sciences, Philadelphia, Pennsylvania, and the United States National Herbarium, Washington, D. C.

Tabulations and discussions of data obtained from this study are here presented, since it is thought that they may be helpful in tracing some of the history and distribution of the disease. A few supplementary data, based mainly on mycological specimens, are included.

HISTORICAL REVIEW OF THE DISEASE

It is generally supposed that the Sphaceloma pathogen of citrus scab, known in the United States as *S. fawcettii* Jenkins, is indigenous to the Orient, where its suscept (Citrus) is (20) also native. According to Tanaka (19), the earliest literature record of the Japanese name of citrus scab is in an unpublished manuscript written in 1818 by Yoshiyuki Murase. This is also the earliest known record of the disease. Other early records in the East are Nagasaki, Japan, 1863 (11), and Queensland, Australia, 1876 (21, p. 4). The Nagasaki record is based (11) on phanerogamic specimens showing scab lesions, in the herbarium of the Hong Kong Botanical Gardens.

In the Western Hemisphere, early North American records of the disease are Florida, 1878 (9), about 1884 (18), and about 1885 (14); and early South American records (on other than the common sweet orange (*Citrus sinensis* Osbeck), Argentina (2) and Paraguay (15), 1920, and Brazil, 1923 (22). Swingle and Webber (18) believed that scab was introduced into Florida on Satsuma (*C. nobilis unshiu* Sw.). If so, such introduction must have taken place between 1876, when Satsuma was first brought to

[1] Part of a paper read at the annual meeting of The American Phytopathological Society, December 30, 1931 (10). The remainder of the paper, dealing with citrus canker, will be presented separately. Thanks are due to A. D. Cotton, W. R. Maxon, C. E. Kobuski, F. W. Pennell, J. Ramsbottom, E. M. Wakefield, and A. C. Weatherby, for courtesies in connection with the examination of the phanerogamic specimens, and to H. R. Fulton and W. T. Swingle for a critical reading of this paper.

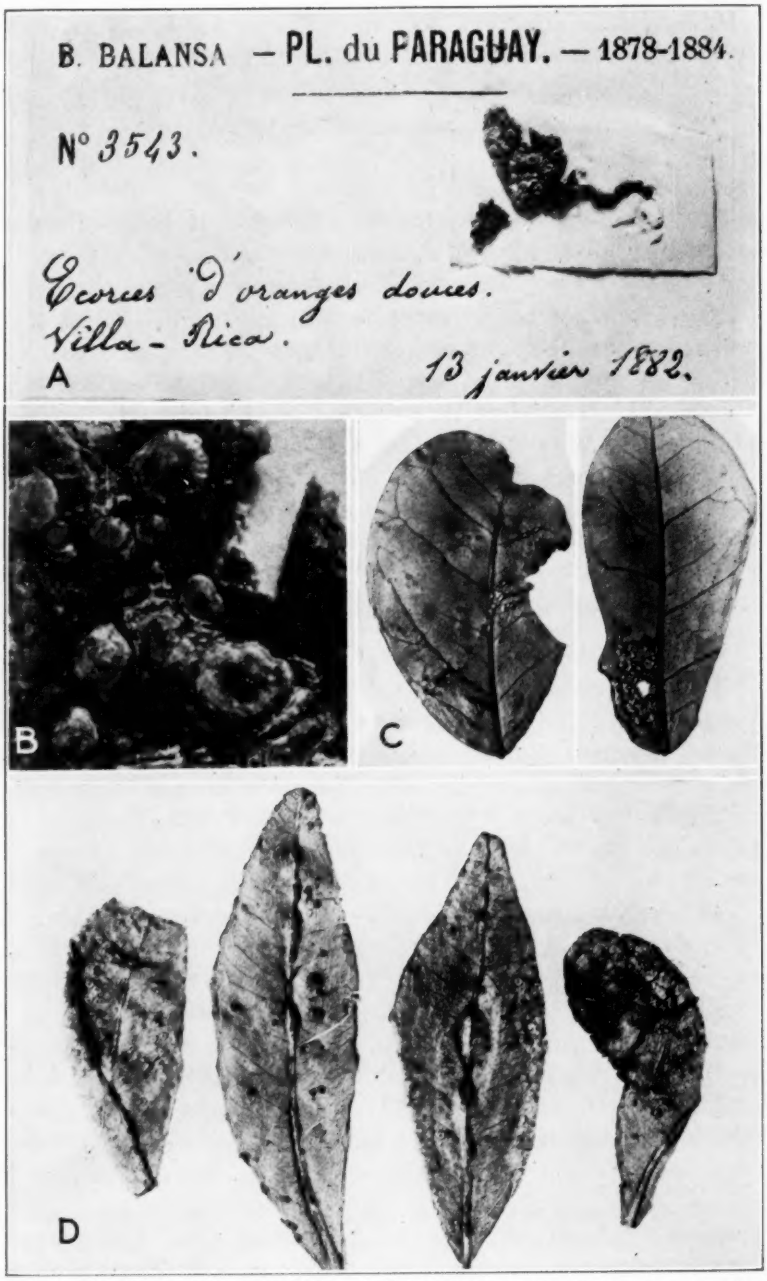

FIG. 1. A. Paraguayan specimen of Sphaceloma on sweet orange, together with label. ×1. B. Enlargement of A. ×7. C. Scab on leaves of *Citrus aurantium*, Nagasaki, Japan, 1863. ×1. D. Scab on leaves of *Citrus* sp. from Chosen, 1906. ×1. Photographs by M. L. F. Foubert.

TABLE 1.—*Identification of citrus scab on phanerogamic specimens of the genus Citrus in herbaria in England in 1930 and in the United States in 1931*

Herbarium[a]	Data on specimens				Name of collection, collector, and number on specimen, where given	Identification
	Date of collection	Place of collection	Kind of citrus			
			Tanaka's identification, where given	Name on label		
Herb. Kew	As early as 1840[b]	Java	*C. limonia* Osbeck	*C. amara* Hassk.	Plantae Bogorienses exsiccatae nova vel minus cognitae H. C. Hochrentiner 107	Apparently scab on leaves and fruit
"	Earlier than 1867[c]	India, Bengal	*C. medica* L.	*C. medica*	Hort. Bot. Calcutta	Apparently affected by scab
"	1862	Japan, Nagasaki	*C. nobilis* Lour.	*Citrus* sp.	Herb. Hook. 1867, Herb. of Royal Gardens, Kew, 134. Coll. R. Oldham	Much scab on leaves and fruit
"	"	"	*C. unshiu* Marcovitsch	*Citrus* sp.	Herb. Hook. 1867. No. 642	Much scab on leaves
Gray Herb.	"	"	*C. aurantium* L.	*Citrus* sp.	Royal Gardens, Kew, 601. Coll. R. Oldham	Typical scab lesions on leaves
Herb. Kew, Gray Herb., and U. S. N. H.	1863	"	*C. unshiu* Marcovitsch	*C. nobilis* Lour.	Ex Herb. horti. bot. Petropolitani Maximowicz Iter secundum Rec'd XII 1885	Typical scab leaf lesions, also funnel-like protuberances characteristic of scab
Herb. B. M., and U. S. N. H.	"	"	*C. aurantium* L.	*C. aurantium* L. var. *bigaradia*	Ex Herb. horti. bot. Petropolitani Maximowicz Iter secundum	Typical scab lesions on leaves (Fig. 1, C)
Herb. B. M.	"	"	*C. aurantium* L.	*C. bigaradia* Duby. var. *japonica*	" "	Much scab on both younger and older leaves
Herb. B. M., and Herb. Kew	"	"	*C. aurantium* L.	*C. bigaradia* Duby.	" "	Apparently affected by scab
Herb. B. M.	1868	India, Bengal	*Citrus medica* L.	*C. limonum* Risso.	Coll. C. B. Clarke	One leaf apparently affected by scab

TABLE 1—(*Continued*)

Herbarium[a]	Data on specimens				Name of collection, collector, and number on specimen, where given	Identification
	Date of collection	Place of collection	Kind of citrus			
			Tanaka's identification, where given	Name on label		
Herb. B. M., and A. A.	1906	Japan, Chosen (Korea)	*Citrus* sp. indet.[d]	*Citrus* sp.	Plantae coreaeae leg. U. Faurie 482[d]	Much scab on leaves (Fig. 1, D)
Herb. B. M.	"	"	*C. tachibunda*	*Citrus* sp.	Plantae coreaeae leg. U. Faurie	Typical scab lesions on leaves
Herb. Kew	1911	"	*C. deliciosa*		Plantae coreaeae	Typical scab lesions
A. A.	1914	Japan, Kogoshima	*C. deliciosa* Kniokuru	*Citrus* sp.	Arnold Arboretum, Exp. to Japan 1914. Coll. E. H. Wilson	Typical scab on leaves
Herb. Kew	"	Japan, Kyushu	*C. tangerina* Hort.	*Citrus* sp.	Arnold Arboretum, Exp. to Japan 1914, No. 6284. Coll. E. H. Wilson	Much scab on leaves
"	"	"	*C. sinensis* Osbeck[e]	*Citrus* sp. "Napura"	Exp. Japan 1914, No. 6825	Much scab
"	1918	Japan, Formosa	*C. tankan* Hayata	*Citrus* sp.	Arnold Arboretum, Exp. to Eastern Asia, 1917–1918. No. 10011. Coll. E. H. Wilson	Typical scab
"	"	"	*C. maxima* (Burm. f.) Merr.		Coll. E. H. Wilson	Typical lessions on leaves
A. A.	1928	China, New Territory		*C. aurantium* ?	Herb. Coll. Agr., Sun Yatsen, Univ. F. No. 624. Coll. W. Y. Chum	Typical lesions on leaves

[a] Herb. Kew = The Herbarium, Royal Botanic Gardens, Kew; Herb. B. M. = Herbarium, Department of Botany, British Museum; Gray Herb. = Gray Herbarium; A. A. = Arnold Arboretum; U. S. N. H. = United States National Herbarium.

[b] The date August 21, 1840, appears at the close of Hasskarl's (7) article in which this specimen is cited.

[c] 1867 = Date this specimen (undated) was given to the Herbarium at Kew.

[d] U. Faurie 482 is identified by Swingle (17) as *Citrus nobilis* var. *deliciosa* Sw. n. comb.

[e] Apparently nearer sour orange (*Citrus aurantium* L.) than *C. sinensis*. H. S. F.

Florida (1), and 1878, the earliest date for the occurrence of the disease there (9).

When the disease was discovered in South America, in 1920, it was thought (2, 15) that it was of recent foreign origin there, although G. L. Fawcett (2) suggested that it might have been present in the country earlier without having been reported. Spegazzini (15) believed that the pathogen had been introduced from Florida. Hauman (8), however, considered that the disease differed in some respects from Massee's (12) description of it, which pertains only to the disease in Florida.

The common sweet orange is generally regarded as immune from or highly resistant to attack by the citrus-scab pathogen. Scab of sweet orange is reported, however, in Paraguay (6, 15), Uruguay (6), and Argentina (3). The mycological specimen collected in Paraguay in 1882 (9) is illustrated in figure 1, A and B. G. L. Fawcett (3), in reporting scab of the common sweet orange in Argentina, says that it is distinct from common citrus scab, affecting sour orange (*Citrus aurantium* Osbeck) and mandarin (*C. nobilis deliciosa* Sw.) only slightly, and being practically limited to fruits. This author[2] has recently said that the disease, which was present in Argentina long before the common scab was introduced, causes a dwarfing of young sweet-orange fruits (less than half grown) and greatly disfigures full-grown fruits. Furthermore, in nonsprayed orchards, of which there are many in Argentina, the disease does not extend through the plantation, but affects only about 5 to 10 per cent of the trees. The name sweet-orange scab is here suggested for this disease.

DATA OBTAINED MAINLY FROM PHANEROGAMIC SPECIMENS

Table 1 lists the phanerogamic specimens on which citrus scab, or what appears to be this disease, was found. It will be noted that all of the material is eastern and that the kinds of citrus represented are those recognized as moderately or highly susceptible to scab. As would be expected, none of the specimens are from the Philippines, where the disease is not known to occur. The diseased phanerogamic specimen from Chosen, dated 1906, showing numerous scab lesions, is probably one of the earliest records if not the first record of the disease in that country. Leaves from the specimen in the Arnold Arboretum are shown in figure 1, D. Assuming that the lesions on the two specimens from Bengal are definitely of scab, these now constitute the earliest known record of the disease in India. The Javanese specimen is the earliest available record of the disease outside of Japan. The only other record from Java is dated 1919 (9).

The prevalence of scab at Nagasaki, Japan, in 1862–1863, on several different kinds of citrus is indicated by the several specimens from Nagasaki listed in the table. Leaves from the specimen in the United States National

[2] Letter dated Dec. 20, 1931.

[d] U. Faurie 482 is identified by Swingle (17) as *Citrus nobilis* var. *deliciosa* Sw. n. comb.
[e] Apparently nearer sour orange (*Citrus aurantium* L.) than *C. sinensis*. H. S. F.

Herbarium, dated 1863, are shown in figure 1, C. The specimen, originally identified as *Citrus nobilis*, dated 1863, is evidently of the same collection as that in the Hong Kong Botanical Gardens, referred to in the previous section. The specimens dated 1862 now supersede this collection as the earliest specimen record of the disease and the earliest record of its occurrence at Nagasaki.

Among the early specimens from the Western Hemisphere examined were those from the British Museum, *i.e.*, sour orange (highly susceptible to scab), from Jamaica (in the Sloane Herbarium) dated 1783, and from the region of the St. John's River, Volusia County, Florida, dated 1848. Upon discussing with Dr. Swingle the fact that this material is scab-free, as is (9) that in the Schweinitz Herbarium, acquired by the Academy of Natural Sciences, Philadelphia, in 1834, also including sour orange, he contributed the information that as late as 1893, when scab was wide-spread in Florida, he found a grove of naturalized sour orange there that was still unaffected. This was in Lake County, on the right bank of Alexander Spring Creek, near its junction with the St. John's River and a few miles south of Dexter's Lake.

Although not found on South American phanerogamic specimens of citrus examined, citrus scab is present on a number of mycological specimens recently received from that continent. The kind of citrus represented and the source of this material are as follows: from Argentina, lemon (*Citrus limonia*) collected in 1925 by G. L. Fawcett and common sweet orange collected in 1927 by Max Kisliuk; and from Brazil, common sweet orange, lemon, and lime (*C. aurantifolia* Sw.) intercepted in 1930–31, by inspectors of the United States Bureau of Plant Quarantine; Satsuma from the Sub-Estacao Experimental de Limeira, Cordeiro (near Limeira), São Paulo, collected in 1931 by Carlos Wright and sent by E. E. Honey, and Satsuma, lemon (Genoa, rough, and sweet), bitter-sweet orange (*C. aurantium* L.), and lime (Rangpur), collected in 1932 at Vicosa, Minas Geraes, by P. H. Rolfs.

The present study gives added support to the idea that scab originated in the Orient at an early date and was introduced into North America from this region. But it affords no explanation of the origin of the form or forms affecting the sweet orange in South America, in one instance known to have been present as early as 1882. This is only 4 years later than the earliest date for scab in the Western Hemisphere and long before scab was found on other kinds of citrus in South America or on sweet orange (16) in North America, *i.e.*, Florida. The investigation adds no new countries to the known distribution of scab on any kind of citrus, unless there is no previous record from Chosen. The combined literature reports of the disease as now available, however, show that its known world range is considerably

greater than in 1921 (13), 1923 (22), or as late as 1926 (5). This range is as follows:

Eastern Hemisphere: Chosen (Korea), Japan, Formosa, China, Siam, India, Ceylon, Malay Peninsula, Java, Oceania (Bonin Islands, Fiji Islands, Guam, and Hawaiian Islands), Australia (Queensland, New South Wales, and Victoria), New Zealand, Africa (Belgian Congo, Nyassaland, Rhodesia, Sierra Leone, and Union of South Africa).

Western Hemisphere: United States (Alabama, Florida, Louisiana, Mississippi, and Texas), Bermuda, West Indies (Cuba, Dominican Republic, Jamaica, Lesser Antilles, Porto Rico), Trinidad, Mexico, Canal Zone, South America (Argentina, Brazil, Colombia, Paraguay, and Uruguay).

Citing Fawcett (4), Peltier and Frederich (13), in discussing the effects of weather on the world distribution of citrus scab, give 23° C. as the maximal temperature at which infection by the scab pathogen may occur. But Fawcett (4) actually obtained infection at 24.5° and at 27.5° C. in an experiment so arranged that the infection period was prolonged (experiment with detached leaves floating in water). It would seem that infection at these higher temperatures should also be considered in studies involving the temperature relations of this organism. Also, the longer infection period at these higher temperatures may indicate that at such temperatures, under natural grove conditions, there would be much less chance for infection than at 16°–23° C. but that infection would not be precluded.

SUMMARY

Data obtained mainly from an examination of phanerogamic herbarium specimens of the genus Citrus in some herbaria in England and the United States support the idea that citrus scab originated and was present in the Orient at an early date. The Javanese specimen dated as early as 1840 constitutes the earliest specimen record of the disease. The two specimens from Bengal (one dated 1868, the other thought to be much earlier) are the earliest specimen records from India and the specimen from Chosen (dated 1906) is probably the earliest, if not the first, record of the disease in that country. The Japanese specimens dated 1862 constitute the earliest specimen records of the disease. Based mainly on combined literature records the present known range of citrus scab, including the form (or forms) affecting the common sweet orange, is given. Attention is called to the fact that Peltier and Frederich, in discussing the effects of weather on the world distribution of citrus scab, do not take into consideration that scab has been obtained experimentally at 24.5° and at 27.5° C. (where the infection period was prolonged) as well as at the range of 16° to 23° C.

BUREAU OF PLANT INDUSTRY, WASHINGTON, D. C.,
AND CITRUS EXPERIMENT STATION
(UNIVERSITY OF CALIFORNIA),
RIVERSIDE, CALIFORNIA.

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PHYTOPATHOLOGICAL NOTES

Physiologic Specialization of Tilletia tritici on Emmer.—Emmer, commonly known as spelt, is grown as a feed crop in the Dakotas and in adjoining states. Vernal (White Spring) or common emmer, now most commonly grown, was brought to the United States by German-Russian immigrants and grown in the Dakotas prior to 1898. Samples of this variety have since been introduced into this region as Yaroslav emmer.[1]

Although the emmers, *Triticum dicoccum*, as a class, formerly were considered resistant to smut, *Tilletia tritici* (Bjerk.) Wint., is now common and frequently destructive in emmer fields of this region. Head infections of 25 to 50 per cent are not uncommon.

Emmer smut caused by *Tilletia levis* Kuehn has not been observed by the writer, although infections by artificial inoculations have been induced without difficulty.

Four collections of *Tilletia tritici* on emmer from different counties in North Dakota and 146 collections on durum wheat were studied critically

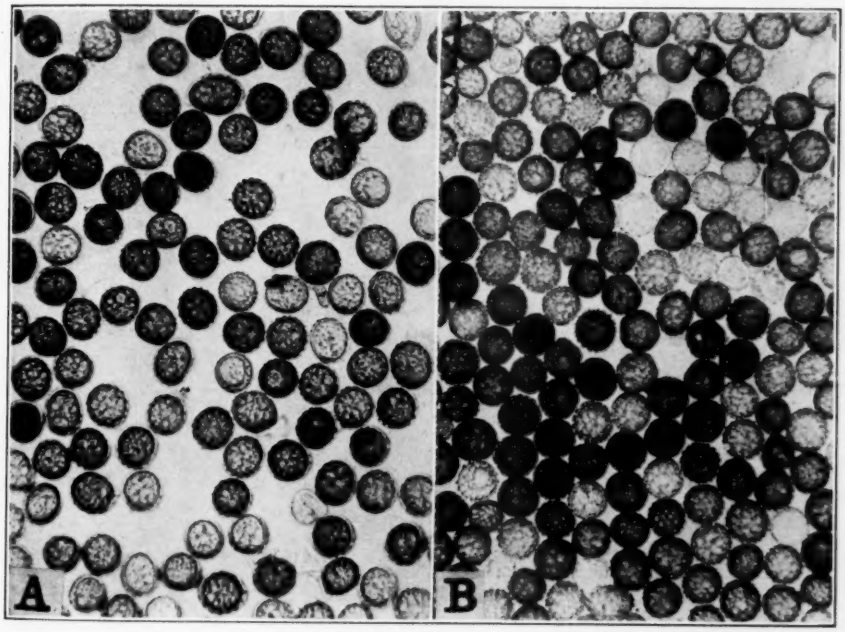

FIG. 1. *Tilletia tritici* from Ceres wheat, developed by artificial inoculations. A. Spores of T408, originating from durum wheat. B. Spores of T394, originating from emmer.

[1] Martin, J. H., and C. E. Leighty. Emmer and spelt. U. S. Dept. Agr. Farmers' Bul. 1429. 1924.

with a microscope. Wide variations were found in the size and shape of spores and in the degree of spore-wall reticulations, but the spores of the 4 collections of emmer smut, T289, T391, T394, and T412, were uniformly unlike these of the durum smuts. They were uniformly of one size, almost spherical and with well-developed reticulations (Fig. 1).

These differences in spores, under certain conditions, were accompanied by a difference in shape of smut balls. This was disclosed by inoculating Ceres wheat.

Single smut balls from each of the emmer collections and from 10 durum collections were each applied separately to 50 seeds of Ceres wheat. The smut balls developing from each inoculation of emmer smut were much elongated, while those from the durum smuts were distinctly rounded (Fig. 2 A). Somewhat similar difference in shape of smut balls from 2 physiologic forms of *Tilletia tritici* was reported by Smith[2] and Holton.[3] In addition to these differences the smut collections were pathogenetically unlike.

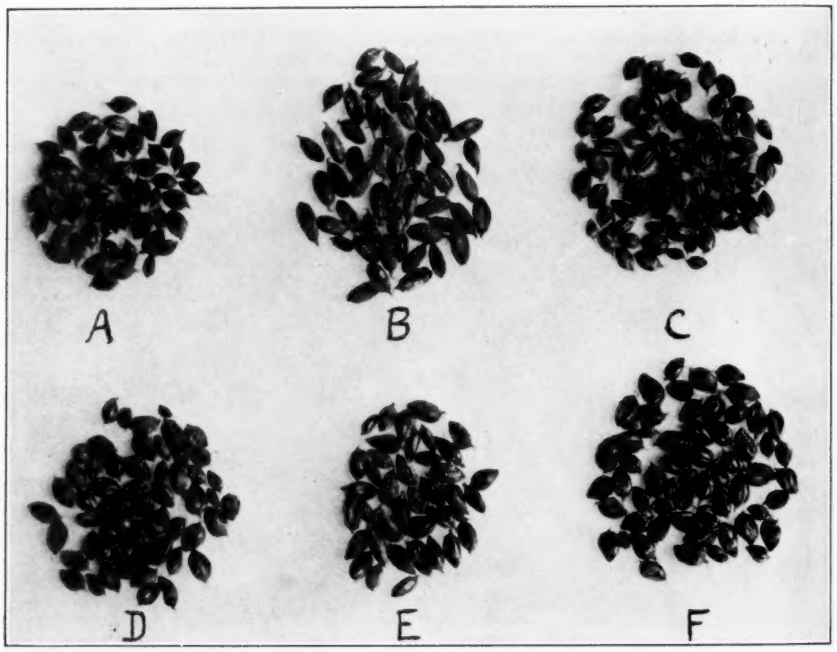

FIG. 2. *Tilletia tritici* from Ceres wheat, developed by artificial inoculations. A, C, D, and F. Smut balls, T401, T402, T405, and T411, originating from durum wheat. B and E. Smut balls, T391 and 394, originating from emmer.

[2] Smith, W. K. Reaction of Martin wheat to three physiologic forms of *Tilletia tritici*. Phytopath. 22: 847–850. 1932.

[3] Holton, C. S. Differences in size and shape of smut balls of two physiologic forms of *Tilletia tritici*. (Abst.) Phytopath. 23: 16. 1933.

In the inoculations of Ceres referred to above the 4 emmer collections produced an average of 24 per cent of smutted heads, whereas the 10 durum smuts produced an average of 86 per cent. In field inoculations, involving triplicated 5-foot rows, the emmer smuts produced low percentages of infection on 10 wheats and relatively high infections on Yaroslav emmer. The 10 durum smuts, conversely, produced relatively high infections on 10 wheats and low infections on Yaroslav emmer.

The collections of emmer smut from the 4 different fields appear to be of 1 physiologic form. Although it is suggested that the emmer smuts of this region generally may belong to this 1 specialized form of *Tilletia tritici*, it remains to be determined definitely whether or not this is true.—W. E. BRENTZEL, Plant Pathologist, North Dakota Agricultural Experiment Station.

Germination of the Oospores of Pseudoperonospora humuli.—Prompt germination of the oospores of the hop disease, downy mildew, *Pseudoperonospora humuli* (Miy. and Tak.), and infection of hop seedlings with inoculum were obtained in connection with trials to determine the disease resistance of new hop varieties. The only other report of oospore germination was made by Arens.[1] His results are substantiated by those here reported, and the importance of the oospore in the study of this destructive disease is established.

Mildew-infected leaves full of oospores, obtained 2 years before, in the fall of 1930, and kept as dry herbarium specimens in the laboratory, were finely ground with a mortar and pestle. The method of germination suggested by Hiura[2] was used. Briefly, the method was as follows: Moist layers of cotton were placed in the upper and lower halves of Petri dishes and a layer of absorbent paper was crumpled to give a corrugated effect on the lower layer of cotton. A small quantity of the powdered leaf was sprinkled on this paper. The contents of the dish were kept moistened and the dishes in all these trials were maintained at room temperature, about 20° C. Germination was obtained also by soaking the mildew-infected leaves in water for several days.

The work was begun on December 20, 1932, and numerous trials were undertaken and observations made several times daily. In a microscopic preparation made from a culture conducted by the first method described and prepared at 11 a.m., December 27, there was evidence of germination

[1] Arens, K. Untersuchungen über *Pseudoperonospora humuli* (Miyabe u. Takah.), den Erreger der neuen Hopfenkrankheit. Phytopath. Ztschr. **1**: 169–193. 1929.

[2] Hiura, Makato. Simple method for the germination of oospores of *Sclerospora graminicola*. Science **72**: 95. 1930.

at 9:30 p.m., on the next day, December 28. Two sporangia had formed and were emitting zoospores, but in the last stages, for the zoospores were moving about feebly and soon rounded up. Most of the zoospores had already escaped, for only 7 were observed to come from one of the sporangia and 8 from the other. Immediately 6 young Late Cluster seedlings growing in the greenhouse were inoculated with this zoospore material. On the first day of January, 1933, there was evidence of conidiophores on 4 of the seedlings, and on January 2 there was unmistakable evidence of mildew infection.

On January 1 some evidence of zoospore formation was found in the leaf material, which had been soaking in water. Eight seedlings of the Fungles variety were inoculated on this date, and on January 4 all of the seedlings were found heavily infected with downy mildew. The seedlings died from the effects of the mildew within a few days.

In subsequent work an excellent section of tissue containing numerous oospores was found in the leaf material soaking in water. Germination was plainly observed in these cases. The oospores germinated by means of a germ tube, perhaps twice the diameter of the oospore in length, which rounded up at its outer extremity into a sporangium similar to the conidia formed in the summer. Germination of this sporangium by zoospores was hastened by slightly warming the glass slide with a lighted match. It was difficult to obtain exact counts, but more than 30 zoospores emerged from each sporangium. Eight seedlings of the Fuggles variety were inoculated with this material on January 18, and on January 21 all of them showed unmistakable mildew infection.

Measurements showed that the oospores were approximately 30 μ in diameter. The sporangia measured about 26 × 31 μ. The zoospores were bean-shape and were 5 to 7 μ in size. The zoospores, after swimming around briskly for about 20 minutes at room temperature, rounded up and came to rest.

Knowledge of oospore germination is not only of value in studying the life history of the fungus but also in controlling the disease. Also, it has been necessary to carry the inoculum on live plants, as this disease is an obligate parasite. Now the problem of carrying the fungus for laboratory and greenhouse study is simplified. For example, the plant breeder may collect oospore material in the fall, germinate the spores when he chooses, and inoculate hop plants. Whether newly formed oospores must go through a dormant period before they are capable of germination is not known. The 2-year-old material used in these trials, however, germinated promptly. Additional studies are being conducted.[3]—E. N. BRESSMAN and R. A.

[3] Published as Technical Paper No. 192 with the approval of the Director of the Oregon Agricultural Experiment Station.

NICHOLS, Oregon State Agricultural College and U. S. Department of Agriculture.

The Spermatia of Flax Rust, Melampsora lini.—*Melampsora lini* (Lk.) Desm. is an autoecious long-cycle rust. It is heterothallic, for an isolated, gametophytic infection cannot proceed to the formation of aecia; it remains haploid. But an infection to which spermatia have been transferred from another infection of opposite sex forms aecia rapidly and discharges aeciospores within 3 or 4 days.

The question arises as to how the spermatia enter and become effective in initiating the sporophyte generation. Although the young flax leaf is tender and the walls of its epidermal cells are relatively thin, few of the rust hyphae of any sort grow out to the surface of the leaf. An intensive search for receptive hyphae reaching the leaf surface between epidermal cells or in stomata gave negative results. The spermogonia offer a feasible avenue of entrance and it is not unlikely that spermatia do grow in through them, but an experiment has proved that spermatia from a mature infection can become effective before the spermogonia of another infection develop. Some other method, therefore, must be available. It would appear that surface hyphae are not essential to fertilization in flax rust.

The extreme minuteness of the spermatia makes difficult a detailed study of their structure and behavior. In infections fixed at short intervals after smearing spermatial exudate on the leaf surface at an infection, the spermatium has been observed to flatten down onto the epidermal wall, secrete minute drops of some substance that may serve to attach it to the wall, and push out a fine beak that penetrates the wall diagonally. There is some evidence that this penetration is effected partly by mechanical force and partly by enzymatic action. The spermatium then enters and moves across the epidermal cell, perhaps carried passively by cytoplasmic streaming. Many die at this stage, perhaps overcome by the defensive forces of the host. There are some indications that a few survive. At the inner wall a spermatium attaches itself, again pushes out a fine beak that passes through the wall and then grows, becoming a hypha within the intercellular spaces.

Cell fusions of paired hyphae are of regular occurrence in the young aecium. This is presumptive evidence that the hyphae of spermatial origin maintain an independent existence up to this point.—RUTH F. ALLEN, Division of Cereal Crops and Diseases, United States Department of Agriculture, cooperating with the Agricultural Experiment Station of the University of California.

A Cork-borer Method for Inoculating Trees.—During the past several seasons the writer has been working with fungus organisms that develop in

the cambial region of true firs (Abies spp.). Various methods were used experimentally to introduce the organisms into the cambium. A cork-borer method of inoculation, suggested by E. P. Meinecke, of the Division of Forest Pathology, has been most satisfactory and is especially adaptable to field conditions.

The method used is as follows: After sterilizing the area of bark where the inoculation is to be made, a $\frac{1}{4}$-inch cork borer is employed to cut through the bark to the xylem. The borer is then carefully withdrawn, containing the bark core in the barrel. The inoculum is deposited in the hole and the core immediately refitted into place by forcing it from the barrel, with the plunger, directly into the hole. The core is secured in place and drying out is prevented by the application of waterproof adhesive tape, which is best wrapped completely around the branch or stem. If the tape is put on in patches it has a tendency to loosen or curl back from the ends and, therefore, seals ineffectively.

This method is particularly useful for trees with relatively thick bark, especially when it is desirable to deposit appreciable amounts of inoculum in the cambial region. When a large number of inoculations are to be made in the field the method as described has the advantages of quickness, simplicity, and effective prevention of desiccation. Serious air contamination is unlikely since the bark core is removed for only an instant while the inoculum is being deposited.—ERNEST WRIGHT, Division of Forest Pathology, U. S. Department of Agriculture, San Francisco, California.

REPORT OF THE TWENTY-FOURTH ANNUAL MEETING OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

THE ATLANTIC CITY MEETING

The twenty-fourth annual meeting of The American Phytopathological Society was held from December 28 to 30, inclusive. Nearly 200 members were present and 112 papers were presented in 11 sessions. There were joint sessions with Section G, A. A. A. S. (the Julius von Sachs Memorial Program) and with the newly formed Mycological Society of America. The contributions presented at this meeting may be classified according to subject as follows: diseases of miscellaneous crops (2 sessions), 15 papers; virus diseases of tobacco, 10 papers; soil treatment, seed treatments, and sprays, 12 papers; diseases of forest trees, 9 papers; virus diseases, 11 papers; diseases of ornamentals, 11 papers; diseases of cereal crops (2 sessions), 20 papers; diseases of deciduous and small fruits, 13 papers; vegetable diseases, 11 papers.

At the annual conference on extension work in plant pathology (held on December 28 under the chairmanship of R. J. Haskell) about 50 specialists took part in discussions of the following topics: use of lantern slides, film strips, motion pictures, photographic enlargements, colored plates, and specimens with demonstrations and exhibits; use of radio (with demonstration), plant-disease schools and study classes, plant clinics, farm-and-home-week programs, contests and clubs, agricultural trains and plant-disease surveys as a method of teaching.

A session on "The Outlook for Plant Pathology under Present Conditions," under the chairmanship of Professor H. H. Whetzel, considered changes in extension methods to meet changed economic conditions and the effect of present economic conditions on the prevalence of some plant diseases.

On December 28 was held the annual dinner, entertainment being furnished under the auspices of the pathologists of New Jersey, New York, Delaware, Pennsylvania, and Harvard University.

Ten new or little-known plant diseases were reported at this meeting: crown gall on *Carnegiea gigantea* (Michael Levine); a sterile but fungus-like leaf spot on pineapple (Melville T. Cook); a new sclerotial fungus from Texas (D. C. Neal and R. W. Wester); a brooming of *Robinia pseudoacacia* (Carl Hartley and L. W. R. Jackson); a Nectria canker of basswood (D. S. Welch); *Nectria coccinea* on beech (John Ehrlich); fig mosaic in California (Ira J. Condit and Wm. T. Horne); a new necrogenic virus disease of potatoes in California (E. S. Schultz and W. P. Raleigh); a Cytospora canker of Picea (C. J. Gilgut and O. C. Boyd). Other scientific contributions included descriptions of the perfect stages of *Sclerotium gladioli* (F. L. Drayton) and of *S. oryzae* (E. C. Tullis); the production of a fertile saltant of *Helminthosporium sativum* by ultraviolet irradiation (F. J. Greaney and J. E. Machacek); acquired immunity to mosaic and spot necrosis in tobacco (J. M. Birkeland); the toxicity of mercuric chloride-potassium iodide solutions (W. P. Raleigh); the genetics of *Sphacelotheca sorghi* and *S. cruenta* (H. A. Rodenhiser); the pathology of crosses between the loose and covered barley-smut fungi (H. A. Rodenhiser and B. F. Barnes); the occurrence of apple scab on stored fruit (C. O. Bratley); the virus diseases of dahlia (Philip Brierley); Sphaceloma on sweet violets (L. M. Massey, R. P. White, and A. E. Jenkins); a new fruit spot associated with apple measles (Anthony Berg); bacterial cankers of Prunus in California (E. E. Wilson); a tuber rot of potatoes caused by *Botrytis cinerea* (Donald Folsom); and a bacterial blight of broad beans in Louisiana (L. H. Person). Abstracts of the papers on the program of this meeting are printed in the January number of PHYTOPATHOLOGY.

OFFICERS AND REPRESENTATIVES

The following officers and representatives were chosen:

President, J. C. Arthur, Purdue University, LaFayette, Indiana.

Vice President, N. E. Stevens, U. S. Department of Agriculture, Washington, D. C.

Councilor (two years), Carl Hartley, U. S. Department of Agriculture, Washington, D. C.

Associate Editors (three years), Lee M. Hutchins, Fort Valley, Georgia; F. L. Drayton, Central Experimental Farm, Ottawa, Canada; Carl D. La Rue, University of Michigan, Ann Arbor, Michigan, and B. F. Lutman, Burlington, Vermont.

Business Manager (one year), F. C. Meier, U. S. Department of Agriculture, Washington, D. C.

Advertising Manager (one year), R. S. Kirby, Pennsylvania State College, State College, Pa.

Committee on Extension (five years), R. E. Vaughan (to replace C. W. Gregory), College of Agriculture, Madison, Wisconsin.

Representatives on the Council of the A. A. A. S. (one year), N. E. Stevens, U. S. Department of Agriculture, Washington, D. C., and H. S. Cunningham, Long Island Vegetable Research Farm, Riverhead, N. Y.

Member of the Board of Governors Crop Protection Institute (3 years), I. E. Melhus, Iowa State Agricultural College, Ames, Iowa.

Representative on Committee on Nomenclature, Botanical Society of America, J. C. Arthur, Purdue University, LaFayette, Indiana.

The following temporary committees were appointed to serve throughout the meetings:

Auditing Committee, Vera K. Charles and C. R. Orton.

Committee on Elections, Mark Woods, A. B. Hatch, and C. S. Holton.

Committee on Resolutions, E. C. Stakman, R. P. White, and W. B. Tisdale.

REPORT OF THE SECRETARY-TREASURER, 1932

At the close of 1931 the Society had 836 members, including the 48 new members elected at the New Orleans meeting. During 1932 six former members were reinstated and a total of 62 members were lost, 2 through death, 18 by resignation, and 42 who were dropped from the Society for nonpayment of dues. Thus, the total membership now stands at 780. Election of the 47 new applicants for membership will bring this number up to 827, a net loss of 9 for the year. Under adverse economic conditions prevailing during the year it would seem that total membership in the Society has been maintained at a surprisingly high figure.

This is especially true in that no drive has been made for new members. It is suggested that members make every effort to call the advantages of membership to the attention of nonmembers who have an interest in the control of plant diseases. Due to the large number of papers presented for publication in PHYTOPATHOLOGY, it will be necessary in the future to accept satisfactory contributions from members only.

STATEMENT OF ACCOUNTS FOR THE YEAR ENDING DECEMBER 14, 1932

Receipts:

Balance from 1931				$2,651.21
Annual dues:	1931	$ 20.00	($10.00 life)	
	1932	1,740.54	(294.53 life)	
	1933	2,009.30	(535.17 life)	
	1934	1.55		3,771.39

Interest on checking account	21.11	
Donations to Lyman Fund included in checks for dues	4.00	
Sales and special sales included in checks for dues	9.63	
Refund by Secretary-Treasurer, expenses of meeting	4.19	
Cash from B. B. Mundkur for dues in other societies	16.17	
Total receipts		$6,477.70

Expenditures:

Member subscriptions transferred to PHYTOPATHOLOGY (1932, part 1931)	$2,813.04	
Transferred to Sinking Fund savings account	886.00	
Secretarial work	333.27	
Printing (programs, preliminary programs, abstracts, ballots, etc.)	159.69	
Advance of funds to Secretary-Treasurer for expenses of New Orleans meeting	125.00	
Expenses of entertainment at New Orleans Phytopathological dinner	35.00	
Mimeographing and mailing press releases	33.84	
Advance arrangements trip to Atlantic City by Secretary-Treasurer	21.80	
Chicago conference of Centennial Exhibits Committee	24.57	
Postage and stamped envelopes	42.36	
Telephone and telegrams	8.76	
Supplies and furniture	63.35	
Express on abstracts and on letterhead paper	2.25	
Visitor's chair and desk chair for Dr. Butler	24.72	
Checks returned by bank	25.00	
Federal tax on checks	.24	
Donations transferred to Lyman Fund	4.00	
Sales and special sales transferred to PHYTOPATHOLOGY	9.63	
B. B. Mundkur, dues in other societies	16.00	
Balance on hand	1,849.18	
Total		$6,477.70

SINKING FUND

The Sinking Fund, obtained by deducting $5.00 (formerly $6.00) from each life-sustaining membership payment, is now $7,663.35, of which $5,000.00 is invested in first-mortgage notes. During the year one note for $1,500.00 was paid up. As recommended by the investment committee the Secretary-Treasurer has opened a savings account with the Riggs National Bank of Washington, D. C. In this account was deposited the above-mentioned $1,500.00 and also $886.00 accumulated during 1931 and 1932, giving a total in this account, with interest, of $2,393.35. (The balance of $270.00 is in the checking account of the Society).

Respectfully submitted,

F. C. MEIER, *Secretary-Treasurer.*

REPORT OF THE BUSINESS MANAGER OF PHYTOPATHOLOGY FOR 1932

STATEMENT OF ACCOUNTS FOR THE YEAR ENDING DECEMBER 14, 1932

Receipts:

Balance for 1931		$1,227.33
Subscriptions: 1930	$ 6.50	
1931	59.99	
1932	2,484.12	
1933	651.91	3,202.52
Sales of back volumes and numbers		202.38
Special half-price sale		673.05
Advertising: 1931	$ 61.56	
1932	537.39	
1933	4.00	602.95
Interest on Sinking Fund		390.00
Principal paid on first-mortgage note		1,500.00
Member subscriptions 1931		25.00
Member subscriptions 1932		2,788.04
Sale of Classics No. 1		2.80
Dues to American Phytopathological Society received with subscriptions		22.50
University of Toronto for share in publishing Forward article		110.00
Palestine Experiment Station for share in cost of reprints		10.00
Total receipts		$10,756.57

Expenditures:

Manufacturing PHYTOPATHOLOGY:			
Vol. XXI, No. 12	$719.02		
Vol. XXI, Index	157.88	$ 876.90	
Vol. XXII, No. 1	621.84		
No. 2	611.36		
No. 3	480.63		
No. 4	769.47		
No. 5	556.60		
No. 6	726.72		
No. 7	347.91		
No. 8	329.17		
No. 9	425.38		
No. 10	506.51		
No. 11	389.57	5,765.16	
Postage		489.21	7,131.27
American Phytopathological Society address list			129.64
Secretarial work for Business Manager			229.85
Expenses of office of Editor in Chief			124.07
Editor in Chief for expenses of New Orleans meeting			60.79

Commission and expenses of former Advertising Manager for 1931	119.45	
Expenses of present Advertising Manager	87.90	
Postage and envelopes	122.15	
Deposited Sinking Fund savings account	1,500.00	
Transferred to American Phytopathological Society for dues	22.50	
Subscriptions refunded	19.85	
Checks returned by bank and collection charge on foreign check	12.50	
Miscellaneous (supplies, reprints, etc.)	12.45	
Federal tax on checks	.36	
Balance on hand	1,183.79	
		$10,756.57

Evidence of the degree to which our Editor in Chief, Dr. H. B. Humphrey, has kept expenses of publication within income is found in the fact that the balance remaining out of 1932 receipts available for publishing the December, 1932, number of PHYTOPATHOLOGY was $527.88. The actual cost of this number was $412.08.

At the end of the calendar year 1931 there were 573 subscribers (nonmembers) to PHYTOPATHOLOGY, including 6 complimentary subscriptions. Fifty-nine (24 from U. S. S. R.) were cancelled for 1932 and during the year 18 were dropped from the list because of failure to maintain their subscriptions. With 40 new subscriptions added during the year the net loss was 37, reducing the subscription list to 536.

Respectfully submitted,

F. C. MEIER, *Business Manager.*

REPORT OF THE EDITOR IN CHIEF

The restrictions placed upon the published output of plant-science journals because of declining revenues have registered their effect also on PHYTOPATHOLOGY. The number of manuscripts submitted during 1932 has, however, furnished no evidence that the general economic depression has been a deterrent to the assembling and preparation of research data for publication. The increasing volume of manuscripts submitted for publication in our journal still bears eloquent testimony to the fact that not a few of the papers fill considerably more space than the substance they present really warrants.

In these times, which call for exercise of the strictest economy, our contributors could do much toward speeding up publication by giving greater care to the preparation of their manuscripts. It is sometimes necessary to return a manuscript to its author two or three times for revision or possible rewriting before it is in acceptable shape for the printer. Many contributors give too little thought to the preparation of tabular data; some of the manuscripts contain tables without headings; others present tables two or more of which could be condensed to a single table. The illustrative matter accompanying the papers published in volume 22 of our journal has, in the main, been more satisfactory than in the past. Some of the illustrations have been exceptionally choice.

The 22nd volume of PHYTOPATHOLOGY contains 1,002 pages of printed matter and illustrations. The preceding volume comprised 1,207 pages of such matter. The 1,002 pages of PHYTOPATHOLOGY for 1932 are classified as follows: seventy-five articles, 30 phytopathological notes, 3 reports, 5 book reviews, 108 abstracts (3 by title only), 1 plate, and 160 text figures. During the period January 1 to December 31, 1932, approximately 157 manuscripts of articles, phytopathological notes, reports, book re-

views, etc., and 136 abstracts (4 by title only) have been submitted. Of this number, 14 manuscripts were returned to their authors for revision, 2 were found unacceptable, and 3 were withdrawn. Of manuscripts submitted in 1931, too late for publication that year, 1 was returned to its author for revision, 2 were not accepted, and 1 was withdrawn. Of the several manuscripts received during the current year, 17 articles, 7 phytopathological notes, and 117 abstracts (2 by title only) are now in press. The index for volume 21 of PHYTOPATHOLOGY was published as a supplement to the February, 1932, number.

The editor wishes to acknowledge with gratitude the highly efficient clerical and editorial service rendered by Frances W. Todd. Thanks are due also to each of the several members of the editorial board and to The Science Press Printing Company for the part each has shared in maintaining the standards of editorial excellence and workmanship of PHYTOPATHOLOGY.

Respectfully submitted,

H. B. HUMPHREY, *Editor in Chief.*

REPORT OF THE ADVERTISING MANAGER

In 1932 a total of 96 advertisements were run in PHYTOPATHOLOGY. These comprised a total of 70⅝ pages. Of the total advertising, the cash advertising consisted of 61 advertisements, occupying 44⅛ pages, valued at $713.05; further, the exchange, PHYTOPATHOLOGY back-number sale, advertising directory, etc., accounted for 35 advertisements, occupying 26½ pages.

The amount and value of the total advertising space contracted in 1932 remained very nearly the same as in 1931; however, PHYTOPATHOLOGY, along with nearly all other journals, was affected by the general business conditions. A total of 19¼ pages of advertisements was lost by cancellation.

An effort has been made during the past year to make PHYTOPATHOLOGY advertising of greater value both to members of the Society and the advertiser. An advertising directory has been included, which lists each advertiser and his products.

The advertising manager wishes to express his appreciation of the valuable assistance and suggestions given by J. F. Adams, the former advertising manager, and to The Science Press Printing Company, publisher of PHYTOPATHOLOGY.

Respectfully submitted,

R. S. KIRBY, *Advertising Manager.*

COMMITTEE OF EDITING PHYTOPATHOLOGICAL ABSTRACTS

That the scientific merit of the abstracts of papers submitted for inclusion in the programs of the annual meetings and for publication in PHYTOPATHOLOGY is still regarded by a considerable proportion of the Society membership as too slight to justify this use of the members' time, the Journal's space, and the Society's funds is well known to the committee on editing abstracts. It is quite patent also that individual members are not disposed to recognize in their own contributions to the Society's abstract problem the same shortcomings as those deplored in the abstracts submitted by others. And some never submit any abstracts at all, so that their proficiency cannot be gauged; but they complain of what other members write, nevertheless. Because the issue of scientific worth of the abstracts is touched with personal interest, the committee still tends to err on the side of toleration. Abstracts are accepted that the committee knows make no significant contribution to our science; the committee is then blamed for low standards and lax enforcement; every year some one proposes that the Society cease printing abstracts, others rise to defend them, "and so, far into the night, the old battle rages."

The real difficulty, as has been pointed out before, is that these abstracts represent a place on the program, as well as the author's conception of a scientific note worthy of publication. The committee on editing abstracts has never received a mandate from the Society to act as a Censor of Programs. It holds, until the Society rules otherwise, that it is the inalienable right of every member to aspire to a place on the program and to submit in fulfillment thereof any title in the field of phytopathology that the Secretary will accept. The substance of the discourse and the manner of presentation are the concern of the individual, subject only to his own regard for his professional standing, the time limit, and the patience of his audience.

Accordingly, this committee does not presume to say what papers may or may not have a place on the program. Our function is construed to be only the reviewing of the abstracts to pass on their scientific merit and novelty, and to edit them when necessary to meet reasonable standards of literary expression. We do not want to enter into a discussion here of the advisability of charging the cost of printing the abstracts against the authors, but we urge the Society to support the abstracts committee, whether this committee flourishes next year under the impetus of a new deal or languishes under its present personnel, in a strict application of the following policy, authority for which was voted by the Society two years ago. Abstracts that do not, in the judgment of the committee, measure up to standards of scientific worth and novelty comparable to those maintained for full-length papers that are printed in PHYTOPATHOLOGY will be published by title only.

Respectfully submitted,

FREEMAN WEISS, *Chairman.*

COMMITTEE ON COOPERATION WITH BIOLOGICAL ABSTRACTS

The editor of BIOLOGICAL ABSTRACTS has proposed that this Society adopt a system, already in effect with several other scientific journals, for more promptly obtaining abstracts of papers published in PHYTOPATHOLOGY. In brief, the system provides for the preparation of abstracts by the author of the paper and sets up a method of distributing requests for abstracts, and in many cases the return of abstracts by authors, simultaneously with the correspondence ordinarily incident to the distribution and return of galley proof.

Consultation with the editorial board of PHYTOPATHOLOGY discloses that the proposed method is acceptable to the editors of our journal and will be given a trial.

According to this system, the editor of BIOLOGICAL ABSTRACTS will furnish the editor of PHYTOPATHOLOGY with a supply of forms for requesting abstracts. One of these requests will be attached to the galley proof of each paper to be published in our journal and will be sent to the author, who will prepare the abstract and return if, with the proof or separately, to the editor of PHYTOPATHOLOGY. The latter will collect all the abstracts that pertain to the original papers, which make up an issue of the journal, and will send them, together with a notice of the probable issue date of this number, to the editor of BIOLOGICAL ABSTRACTS. As soon as this number of the journal is distributed, the editor of BIOLOGICAL ABSTRACTS will send the abstracts pertaining thereto to his assistant editors in charge of the respective sections. Thus, all lapse of time between the appearance of the original paper and the securing and distribution of abstracts to the section editors of BIOLOGICAL ABSTRACTS will be avoided. It is expected that the abstracts may even be published within a month or two of the publication of the original paper.

It is hoped in this way to make BIOLOGICAL ABSTRACTS of real service as a current review of original publications within even a limited field, such as phytopa-

thology, in addition to its service in covering the biological field as a whole and in furnishing an annual subject-matter index thereto.

Respectfully submitted,

FREEMAN WEISS, *Acting for the Chairman.*

REPORT OF COMMITTEE ON INVESTIGATION OF FOREIGN PESTS AND DISEASES

An expedition sent by the Bureau of Plant Industry (Horticulture and Foreign Plant Introduction) to South America discovered potato wart in Bolivia, thus establishing for the first time the presence of this disease in that country, and, incidentally, throwing further doubt upon the European origin of *Synchytrium endobioticum*, since this fungus has been known from Peru since 1929.

A little-known smut of potatoes caused by *Polysaccopsis hieronymi* (Schroet.) P. Henn. was collected in the Andes Mountains near Meridia, Venezuela, by C. E. Chardon and forwarded to Washington for identification. This is the first report of this smut on potato tubers, the former collections from Argentina, Bolivia, and Brazil being recorded as occurring on branches and pedicels of *Solanum* sp. It occurs in Stevenson's list under the name *Urocystis hieronymi.*

The smut disease of Colchicum bulbs was intercepted in 1932 at Washington in shipments from Holland. As high as 12 per cent of the bulbs were infected with *Urocystis colchici* and higher percentages were detected in 1931 shipments.

In view of the present conditions with respect to funds for our investigations in foreign countries, the following suggestions are offered for consideration:

(1) Members of our Society who are making visits to foreign countries might secure valuable information with respect to specific parasites if they would communicate with the committee concerning their itinerary.

(2) Similar visits by mycologists, botanists, horticulturists, and others familiar with pest problems could be made contributory to our knowledge of foreign-pest and disease control. The committee will welcome such cooperation.

(3) In view of the financial difficulties already mentioned, there is another avenue of help, *i.e.*, the assistance of special *attachées* of the Diplomatic Service. Such assistance might be helpful in many ways and should be carefully considered.

C. R. ORTON, *Chairman*,
W. H. MCCUBBIN,
F. C. FROMME.

COMMITTEE ON PRESS

Following the custom of the past 4 years, the press committee placed its duties in the hands of the Press Service of the The American Phytopathological Society, an independent organization established for the purpose of issuing the abstracts of the midwinter meetings in a form suitable for publication in the newspapers of the country.

The abstracts (with the exception of about 5 were rewritten and 50 mimeographed copies released through Austin Clark. In previous years, it will be recalled, between 25 and 30 pages of mimeographed news stories have been released to nearly 500 newspapers and farm magazines. This year it has not been possible to continue this work.

That this work has been profitable and well worth the Society's efforts is evidenced by the response from the public to a story on the use of naphthalene (*cf.*, Weiss & Evinger, PHYTOPATHOLOGY **22**: 1932) as a fungicide given wide circulation *via* the NEW YORK TIMES, as well as the accounts published in the MARKET GROWERS JOURNAL wherein nearly all the vegetable papers of last year were reported.

It is hoped that in another year, if this sort of beneficial press agentry be continued, a greater degree of cooperation between the membership of the Society and various institutions may be maintained, so that the material can be in the press committee's hands as soon before November 15 as possible, in order that it may be assembled in its effective form, mimeographed, and mailed not later than December 1.

For his invaluable suggestions this year, as in earlier ones, the committee is indeed grateful to Mr. Don B. Reed, managing editor of the WASHINGTON POST. Mr. Reed, in his capacity as consulting editor of the Press Service, especially last year (1931), was unquestionably responsible for the hearty reception given our material by the press of the country.

W. A. WHITNEY, *Chairman.*

COMMITTEE ON ELECTIONS

The committee on elections, after having opened sealed ballot envelopes and counting ballots for each nominee, reports election of the following officers: President, J. C. Arthur, Vice President, N. E. Stevens, Councilor, F. D. Heald.[1]

MARK WOODS, *Chairman.*
A. B. HATCH,
C. S. HOLTON.

COMMITTEE ON NECROLOGY

During the calendar year 1931, there have been two deaths, as follows:

Prof. Arthur Jaczewski, February 12, 1932.
Dr. Roland Thaxter, April 22, 1932.

A. G. JOHNSON, *Chairman,*
G. P. CLINTON,
M. B. WAITE.

REPORTS OF OTHER COMMITTEES AND REPRESENTATIVES

American Association for the Advancement of Science. A brief, informal report of the representatives on the *Council of the American Association for the Advancement of Science* was presented by D. Reddick.

Auditing Committee. The Auditing Committee appointed December 28, has examined the books of The American Phytopathological Society and PHYTOPATHOLOGY and found them correct.

VERA K. CHARLES, *Chairman,*
C. R. ORTON.

Committee on Extension Work in Plant Pathology. The activities of the committee on extension work have been chiefly along two lines: the publication of the mimeographed leaflet *The Extension Pathologist* and the making of arrangements for the extension conference at the present Atlantic City meeting. Owing to the fact that funds for the mimeographing in the U. S. Department of Agriculture were cut in half this year, only 3 numbers of *The Extension Pathologist* have been issued to date and each number is restricted to a total of 8 mimeographed pages. The committee will appreciate receiving suggestions for improvement and also contributions to this publication.

No summer meetings of the committee or of extension pathologists were arranged in 1932 because of the unsettled economic conditions.

C. T. GREGORY, *Chairman.*

[1] See item under Other Business.

Committee on Century of Progress Exposition. At the request of the Chairman, L. R. Jones, the duties of this office were assumed in 1932 by H. W. Anderson. A meeting of a part of the committee was held at Chicago, where contact was made with J. F. W. Pearson, who has charge of the exhibits on biologic science in the Basic Science Building. The plans of the committee called for financial support from the general funds of the Century of Progress Exposition. According to Dr. Pearson, this support was not possible because of the lack of funds. He gave us to understand that funds for other exhibits were being supplied from commercial sources. It was the opinion of the committee that we were not empowered to go ahead under these conditions, and so reported to the Secretary of the Society.

In correspondence with Secretary Meier it was suggested that the committee request support from the Agricultural Section as represented by A. F. Woods and C. W. Warburton of the United States Department of Agriculture. It seemed from their statements that sufficient financial support could not be secured to warrant a special exhibit, which, in the opinion of Dr. Pearson, would cost in the neighborhood of $5,000.

It is the opinion of the Acting Chairman that your committee should continue to function in an advisory capacity but should make no further effort to secure a separate phytopathological exhibit.

(Dr. Jones requests that H. W. Anderson be made active Chairman of the committee.)

L. R. JONES, *Chairman*,
C. T. GREGORY,
G. K. K. LINK,
M. B. WAITE,
H. W. ANDERSON.

Representatives on Board of Governors, Crop Protection Institute. The Crop Protection Institute continued its productive work during 1932 with as many active research projects as in any other year of its history. The scope of its work is gradually increasing, as more and more industrial organizations entrust it with funds for prosecution of research. Large corporations have reduced their research activities and it has been difficult throughout 1932 to secure financial support of research by industrial funds. Corporations have been interested in arranging for preliminary exploration during this period of reduced business activity. They are interested in searching for new outlets and new products at the present time, so they may be in a position to engage in more comprehensive development later and may have a groundwork laid for such plans. Preliminary exploration of this kind can be done at moderate expense and can be carried along step by step over relatively short periods of obligation.

In 1932 the Institute directed 16 active research projects. Eight of these projects were related to problems in plant pathology, covering such phases as development of new copper sprays and dusts, iodine salts as fungicides, fungicides in combination with oil sprays, combined fungicides and insecticides, industrial adhesive tape to reduce losses from crown gall, plant introduction and improvement, development of flotation sulphur, copper salts in relation to plant nutrition and plant stimulation, etc. The various projects are being conducted through the cooperation of 9 experiment stations, and each project is under the supervision of a committee composed of 3 plant pathologists. Publication of results has been made where conditions warrant publication.

The Board of Governors, as in the past, is composed of 3 members elected by the American Phytopathological Society, 3 members elected by the American Association of Economic Entomologists, and 2 members elected by the Association of Agricultural Chemists. The officers elected by the Boards of Governors for 1933 are W. C. O'Kane,

Chairman, Durham, New Hampshire; J. F. Adams, Vice Chairman, Newark, Delaware, and Paul Moore, Secretary and Treasurer, National Research Council, Washington, D. C.

W. F. MARTIN, *Chairman*,
J. F. ADAMS.

Resolutions Committee. The American Phytopathological Society wishes to express its heartiest thanks to Albert Skean, Executive Secretary of the Atlantic City Convention and Publicity Bureau, for his very efficient help in making such satisfactory arrangements for the meetings of the Society.

The American Phytopathological Society wishes to express its appreciation to the management of The Madison Hotel for extraordinary courtesies and hospitality during the meetings. We wish to thank especially Frank E. Sutch, Publicity and Convention Manager, Eugene C. Fetter, Managing Director, and Linton B. Arnold, for extraordinary helpfulness in connection with our meetings.

The generosity of Domenico Pirone has made it possible to translate and make available to pathologists and mycologists the extremely interesting publication of Fontana on rusts. The American Phytopathological Society wishes, therefore, to express to Mr. Pirone its appreciation of his interest in the progress of our science and its deepest gratitude for his extraordinary generosity. The Society is thankful also to P. P. Pirone for making the translation.

Whereas the committee on the banquet program labored very effectively, possibly even long and arduously, in delineating our individual and collective mental and emotional growth curves, much to our profit and merriment, *BE IT THEREFORE RESOLVED* by The American Phytopathological Society, even after having recovered from the immediate effects of the meteoric shower of pungent wit, that we express our appreciation of the kindly spirit in which the quips were quipped and our thanks for the thought- and mirth-provoking efforts of W. H. Martin, Chairman of the committee, to Richard White, to Mr. Ology, McGinty, and others who helped produce the festival of fun.

Recognizing the fact that the Secretary-Treasurer of the Society and Business Manager of PHYTOPATHOLOGY, F. C. Meier, and the Editor in Chief of PHYTOPATHOLOGY, H. B. Humphrey, work hard and get in return nothing except occasional criticisms and complaints, we wish to record our appreciation of their efficient and unselfish services and to thank them deeply and sincerely for contributing their time and energy to the affairs of the Society.

BE IT RESOLVED that no limitation be placed on the surge of New Year's resolutions to help insure the future financial support for our journal, PHYTOPATHOLOGY, of which we are all proud and of which we wish to become prouder, and that we resolve to transmutate our resolutions into donations, in order that we may help the present generation and future generations to publish promptly and effectively the results of pathological researches.

E. C. STAKMAN, *Chairman*,
W. B. TISDALE,
RICHARD WHITE.

ACTION OF THE COUNCIL

In addition to making the appointments of the officers mentioned earlier in this report, the Council submitted the following actions, which were approved by the Society.

1. On consideration of an offer from H. H. Whetzel to personally handle for the Society business management of Phytopathological Classics, with editorial work by H. B. Humphrey, it is recommended that this offer be accepted with the provision that the arrangement may be terminated at the will of either party on 1 year's notice, the money on hand and remaining stock to be returned to the Society.

2. It is recommended that, in conjunction with the general meeting of the American Association for the Advancement of Science and associated societies, a combined Chicago and summer field meeting be scheduled during the time set by the American Association for the Advancement of Science and that the President advise with the Council regarding appointment of a committee to handle arrangements.

3. It is recommended that enrollment of new life members be temporarily discontinued, pending study by the committee on investments of the financial return from this class of members, the committee, if necessary, to increase the rate for new members in this class.

4. On consideration of the excellent service to the Society rendered by W. A. Whitney, who, for several years, has served as press representative, the Council passed a resolution commending Mr. Whitney for this work that urged him to reconsider his resignation from these duties. The Council instructed the Secretary to report on Mr. Whitney's work in some detail to the Society in business session.

5. That, as was the case in 1932, the Editor in Chief of PHYTOPATHOLOGY be allowed actual expenses for 1933 up to or within $300.00 for secretarial and editorial assistance.

OTHER BUSINESS

The reports of officers, committees, and representatives, as printed above, were accepted.

The Secretary read the following from a telegram from President F. D. Heald—"Prevented from coming to Atlantic City by influenza. Greatly disappointed as I had been looking forward to the trip and the pleasure of meeting many old friends and making new acquaintances. Best wishes for an enjoyable and successful meeting." Following this reading, the Society instructed the Resolutions Committee to prepare a telegram to be sent by the Secretary expressing regret of those present in Atlantic City at President Heald's enforced absence.

It was moved and carried that in view of the fact that under the Constitution the retiring President, F. D. Heald, automatically retains a seat in the Council for 1933, Carl Hartley, who received the second largest number of votes in the mail ballot, be elected Councilor for 2 years.

Resignation of L. R. Jones from the Committee on Exhibits at the Chicago Century of Progress Exposition and his recommendation that H. W. Anderson be assigned the chairmanship of this committee were approved.

Attention was called by the Secretary to good results obtained from activities of press representative W. A. Whitney and methods used in making his popular rewrites of abstracts available to the press.

Proposal submitted in a telegram by W. A. Whitney to the effect that the annual meetings in 1933 be devoted to a Silver Anniversary and Memorial Program was referred to the Council with power to act.

Motion made by J. C. Walker that the Council, in consultation with the Editor in Chief and the Business Manager of PHYTOPATHOLOGY, be empowered to charge authors a set price per page for articles published in PHYTOPATHOLOGY, said price to be determined by the Council, provided said price does not exceed $1.00 per page, was carried.

Motion introduced by A. G. Johnson that publication of abstracts be abolished was defeated after much discussion.

It was moved by L. R. Jones and carried that the Council be empowered, in the case of abstracts, to charge up to full cost of publication, removing the limit of $1.00 per page previously authorized for publications in PHYTOPATHOLOGY.

ARTHUR JACZEWSKI

NOVEMBER 3, 1863–FEBRUARY 12, 1932

Arthur Jaczewski was privately educated in Russia and took advanced studies in the University of Berne, Switzerland. He also attended special lectures at the Sorbonne and the universities of Lausanne and Montpellier.

In his earlier years he botanized extensively, privately, in Russia, Switzerland, Italy, and Algeria. In 1901, he founded the "Central Laboratory of Plant Pathology" and was its director until 1905. In 1907, he became the director of the newly organized "Bureau of Mycology and Phytopathology," which, in 1917, was reorganized into the Division of Mycology and Plant Pathology. Later this was elevated to a national "Institute," named for him, "Institut Jaczewski."

Professor Jaczewski was an unusually capable, productive worker, publishing much in the field of mycology and plant pathology. He was dominated to the last by worthy ideals of service through science, giving all he had in zealous pursuit of these, undaunted, undistraught, let happen what might. And withal, as his intimate associates and family knew him, ever the same courteous considerate gentleman; pure-minded, open-hearted, honest seeker of right and truth, interested in art, music, and all good things; "kind and amiable to everyone," "respected, admired, and loved by friends, associates, and pupils."

ROLAND THAXTER

AUGUST 28, 1858–APRIL 22, 1932

Roland Thaxter, in preparation for his life work, studied at Harvard University, receiving the A.B. degree Magna Cum Laude in 1882, devoted two years to work in the Harvard Medical School, and returned to graduate work under the Harris Fellowship to specialize in mycology and cryptogamic botany with Dr. Farlow, receiving the degrees of M.A. and Ph.D. in Natural History in 1888.

From 1888 to 1891 he served as Mycologist at the Connecticut Agricultural Experiment Station in New Haven, carrying on important pioneer work in the study and control of plant diseases, yet still continuing his activities in pure mycology. In 1891 he was called back to an assistant professorship as Dr. Farlow's associate, and in 1896 was given the full responsibilities of teaching and research in cryptogamic botany when Dr. Farlow relinquished all instruction save the guidance of occasional research students. In 1901 he became full professor, the position he occupied until 1919, when he became professor emeritus and honorary curator of the Farlow Library and Herbarium.

His publications comprise a list of 88 papers, chiefly concerned with fungi, but to some extent dealing with other groups of cryptogams and even with insects. The five volumes of his monograph of the Laboulbeniales stand out as one of the greatest of works in the history of mycology.

He exerted a distinct influence on plant pathology through his own brief but outstanding work in this field, through his inspiring example of the accuracy and completeness of his investigations, through the aid he gave to pathologists who consulted him in difficult mycological problems, and through the training and ideals he transmitted to students. In the course of his work he traveled extensively and made valuable and unusual collections. His eminence was internationally recognized by honorary memberships in learned societies and academies, by prizes, and by election to positions of honor in important societies. He was American editor for the Annals of Botany from 1907 until his death.

An outstanding scholar and investigator, an effective teacher, his nearly fifty years of devoted work exerted a significant influence far beyond his own sphere of endeavor, and his death was a great loss not only to mycology but to plant pathology as well.